THE RELATIONSHIP BETWEEN PERSONALITY AND CAFFEINE INTAKE ON VARIOUS PHYSIOLOGICAL AND PSYCHOLOGICAL RESPONSES

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ABSTRACT

A survey and an inventory were administered to investigate the relationship between caffeine ingestion and personality attributes of the 34 student volunteers. The research was carried out in two parts. Firstly, average personal doses, as determined by the survey, were administered and a reaction time task occurred. Blood pressure, heart rate and performance on various mood scales were obtained. The doses were increased on two successive sessions by increments of 50 percent and responses were monitored pre and post caffeine.

Secondly, individuals received the dose of caffeine at which they had previously performed best on the reaction time task in experiment one. These individuals were then compared on a second reaction time task with control group members.

This research incorporated the Yerkes-Dodson Curvulinear Theory of Arousal and Eysenck's Personality Theory in an attempt to further understand the introversion-extroversion dimension with respect to arousal. Unlike other studies in the area, an attempt was made to individualize the doses of caffeine administered so as to clarify the level at which arousal patterns, as determined by the parameters used, varied between the two personality types.

There was limited uniformity in the results obtained from both analyses and there were also some

results contrary to expectations. The findings on one analysis implied a poorer performance by introverts when compared with extroverts on the reaction time task in the pre-caffeine phase. Also, contrary to expectation, introverts performed best in the pre-post caffeine phase at doses which exceeded optimal doses for extroverts and no significant results were obtained for reaction time in a second analysis.

There were numerous inconsistencies in the results which incited a questioning of the validity of the introversion-extroversion dimension of the Eysenck Personality Theory. It is suggested that variables such as subject sensitivity and masking effects of caffeine may be partially responsible for the inconsistencies which infiltrate the caffeine literature, to which this research now makes its contribution.

CHAPTER I

INTRODUCTION

1. GENERAL INTRODUCTION

The basis of this research is the observation of discrepant findings in the literature regarding the effects of caffeine on reaction time. As has previously been discussed, several r searchers have proposed reaction time on specific tasks to be a useful parameter in the investigation of caffeine (Goodman & Gilman, 1980; Hollingsworth, 1912; Nash, 1962; Osborne & Rogers, 1985).

Osborne and Rogers (1985) suggested that reaction time could be expected to decrease at doses of between 80-200 mg of caffeine. This estimate has been mirrored to an extent in the research of others, however it lacks specificity. This is a feature which dominates the caffeine literature and makes direct comparison between studies virtually impossible. This is especially the case when the effects of caffeine are investigated between studies in which the tasks selected have no relation to one another. Although caffeine has been shown repeatedly to decrease reaction time, Carpenter (1962) argued that this may not be the case in tasks requiring delicate muscular movements.

Research into the effects of caffeine is also often confounded by ethical and methodological problems. For

example, it is not ethical to administer near toxic doses to volunteer subjects. There appears to be a wide amount of variance between studies in the amounts of caffeine contained in an average 150 mg cup. Dalby (1985) suggested 142 mg/150 mg of caffeine in percolated coffee when compared with Sawyer, Julia and Turin (1982) who suggested brewed coffee contains 85 mg/150 ml. Greden (1974) and Greden, Victor, Fontain and Lubetsky (1980) suggested that a 5 oz cup of coffee contains 100-150 mg of caffeine. These variations not only apply to coffee but also to tea and carbonated drinks. Srisuphan and Bracken (1986) suggested between 29-176 mg/cup of caffeine in coffee while between 8-107 mg/cup of tea. When there are such wide discrepancies in the amounts of caffeine, this suggests that any result obtained must be treated with scepticism, set aside from any other variable under investigation.

In view of the broad approximations of caffeine content in tea, coffee and carbonated drinks, there is also a broad range of reaction times found both within and between populations. Research has shown that when a population ingests a predetermined uniform dose, instead of obtaining uniform changes in task reaction time and in physiological measures, the opposite occurs (Schilling, 1921). Some of these effects can be explained by virtue of recent food consumption, fatigue, body weight and presence of other stimulants or depressant agents. However, this poses questions regarding whether these

variables exert influence significant enough to account for all inconsistencies in reaction time. In partial explanation to this problem, methodological shortcomings in many cases may have been responsible. Often crossover designs are used and although efficient, they lack the validity present in double blind experiments.

One major methodological flaw is that often the sample population can not be considered representative of the greater population, such as the Weathersbee, Olsen and Lodge (1977) study. A second major problem is that subject recruitment may be dependant on psychological diagnosis. For example, many studies have been carried out to investigate caffeine consumption among individuals with anxiety disorder (Downing & Rickels, 1981; Freitas & Schwarts, 1979; Greden, Fontain, Lubetsky & Chamberlain, 1978; Greden, 1974; Lee, Cameron & Greden, 1985; Lutz, 1985; Sawyer et al., 1982; Winstead, 1976). One would expect the diagnosis of anxiety disorder to be uniform, but in fact this often is not the case and in some instances there is no information provided regarding how the diagnosis of anxiety disorder was arrived at (Downing & Rickels, 1981). Contrary to this, however, some studies offer much information regarding diagnosis (Greden et al., 1978; Lee et al., 1985; Winstead, 1976).

Although methodological shortcomings partially explain observed inconsistencies in reaction time and in various physiological measures, they can not be used to describe these phenomena in full. If inconsistencies in

the caffeine research are to be explained, it may be essential to investigate various attributes existing in the subject population which could account for these differences.

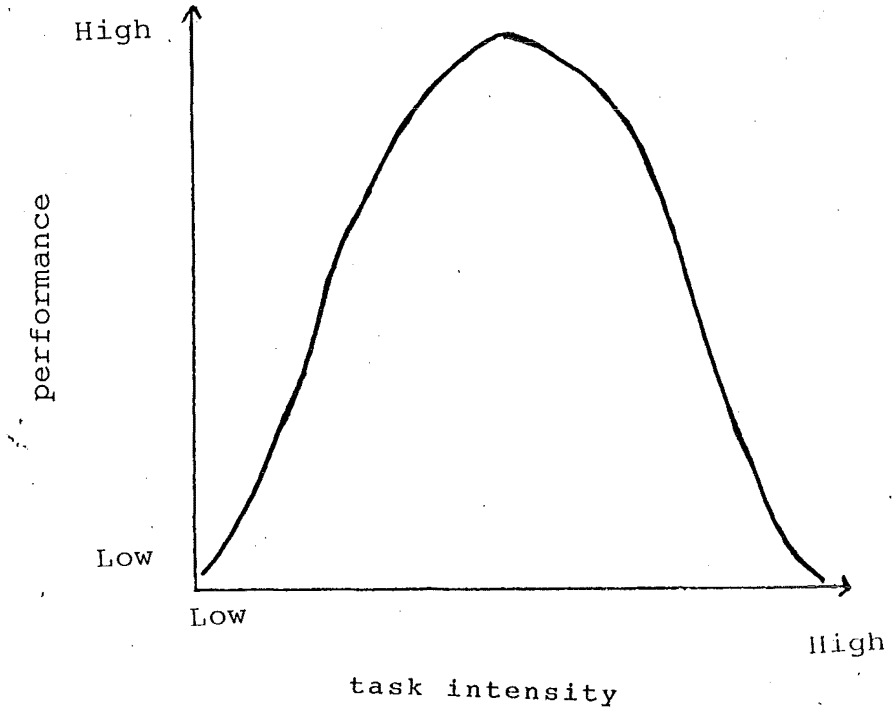
As was previously mentioned, much research has been carried out regarding the interplay between various personality attributes and caffeine ingestion. It has already been established that caffeine has the propensity to decrease reaction time. It is now a question of the attributes of individuals which determine the degree of reaction time change which occurs. Perhaps the most influential research in this area was that which was carried out by Eysenck and culminated in the Eysenck Personality Inventory (1963). This heralded extensive research on two personality attributes, namely (1) Introversion; and (2) Extroversion.

Eysenck based this distinction on the assumption that the two personality types responded to environmental stimuli of equivalent strengths, in different physiological ways. He affirmed that the differences in responding between the two groups were due to differences in the curvulinear relationship between arousal and performance which were unique to each. That is to say that introverts demonstrate higher levels of arousal than extroverts. In light of the curvulinear tneory of arousal (Yerkes & Dodson, 1980), caffeine should promote changed responses between introverts and extroverts and that, depending on the complexity of the task performed, there will be

differences in the optimal level of arousal between the two personality groups. The Yerkes-Dodson theory (1980) asserted that human arousal follows a curvulinear function, that is to say, that as arousal increases, performance improves until a threshold or optimal performance (arousal) level is reached. Once this threshold is surpassed, decrements in performance are observed.

Broadbent (1959) suggested that it is overarousal rather than underarousal which interferes with performance. This assumption is probably based on the observation that overarousal is easily recognisable through symptoms such as nausea, vomiting, convulsion, insomnia and wakefulness, whereas the symptoms of underarousal are more subtle. However, this assumption is dispelled by the theory under discussion. The Yerkes-Dodson Curvulinear Theory of Arousal incorporates both over- and underarousal to explain the variation between optimal and suboptimal performance. Diagrammatically, curvulinear arousal can be summarized in the following way:

Illustration 1: The Yerkes-Dodson Curvulinear Theory of Arousal (1908)



Easterbrook (1959) expanded on this theory by hypothesising that performance can be enhanced if individuals are performing tasks requiring minimal cognitive processing. This hypothesis suggests that individuals have the capacity to determine those features which are fundamental to task performance and to direct attention to these rather than those factors superfluous to the task. If this were not the case, the process would be grossly inefficient and arousal levels considerably lower. It is conceivable that differences in method of response made by introverts and extroverts in view of this single attribute could account for the differences in arousal in which, according to Eysenck, introverts require less arousal than extroverts.

It is also appropriate to incorporate a discussion

of task complexity at thi point. It has been documented by several researchers that this factor can contribute greatly to fluctuations in arousal over the course of the day. Eysenck and Folkard (1980) and Hamilton, Hockey and Quinn (1972) suggested an improvement in those tasks requiring limited storage and rapid processing as the day progressed while Hockey, Davis and Gray (1972) stated that impairment occurred over the course of the day for tasks employing short-term memory. This may provide one method of clarifying any existing controversy regarding the predictive qualities of the Introversion/Extroversion distinction in Eysenck's Personality Inventory.

There is limited p ysiological strength suggestive of a role played by personality in relation to caffeine induced responses. Caffeine has been shown to be an adenosine antagonist. That is to say, caffeine inhibits binding at adenosine receptor sites and, according to Charney, Galloway and Heninger, (1984), this results in an increase in stimulation which may manifest itself through overt behaviour such as diminished reaction time and through physiological parameters such as pulse and blood pressure.

The personality centres which have historically been sited in the frontal lobe share a close proximity to that area most affected by caffeine, namely the cortex, which comprises thalamic nuclei projections which receive most neural input. Consequently, it would not be unreasonable to hypothesise some formal link between adenosine

receptors and personality centres. Extensive additional investigation may yield more decisive findings on the physiological aspects of personality and its role in reaction time tasks dependent on caffeine.

Despite the vast amounts of research carried out on arousal and more precisely arousal and personality, and the challenges made to existing theory, Eysenck's theory of arousal based on the research of Yerkes and Dodson (1908) remains the most influential. It provides a highly regarded framework to test and evaluate arousal on specific tasks. As Revelle, Humphreys, Simon and Gilliland (1980) stated, the theory is largely descriptive rather than explanatory. However, this must be regarded as a strength because it facilitates research on related factors which may extend the theory at some later time. For this reason, it is the framework adopted in this research.

Caffeine was selected for study because it represents one of the most widely used psychoactive drugs in existence today and because its use promotes both observable behavioural effects and cognitive effects. A second reason for investigating the effects of caffeine was that although numerous studies have been carried out in this area, there are often shortcomings such as lack of subject representativeness in terms of subject sensitivity and normal consumption levels. It is intended that this research may contribute to the clinically significant information already available regarding the relationship between caffeine, personality and reaction-time. A third

consideration in the investigation of caffeine was to minimize the range of administered doses in an attempt to establish more precision so that comparisons between groups may be strengthened.

2. RATIONALE

This research was carried out in order to identify the changes instigated within a population of introverts and extroverts as a result of caffeine ingestion at doses predetermined as appropriate for each individual. The ten independent variables of concern were reaction time, systolic blood pressure, diastolic blood pressure, heart rate and several mood characteristics comprising relaxed, normal breathing, sleepy, unhappy, calm, confidence lacking, and their opposites. These variables were measured prior to caffeine ingestion and following caffeine ingestion.

They were considered because they have dominated the caffeine literature for many years and because investigation may provide an insight as to the effects of caffeine on these aspects of human functioning.

The general rationale adopted in this research was to test the validity of the Yerkes-Dodson Curvulinear Theory of Arousal (1908) and subsequently the validity of the Eysenck Personality Theory of Arousal by using a simple reaction time task, so defined because it required minimal cognitive processing in order for the subject to

make the appropriate response. Under the Curvulinear Theory of Arousal, introverts and extroverts, as determined by the Eysenck Personality Inventory (1963) were expected to perform differently at manipulated levels of caffeine. As the caffeine levels increased, introverts were expected to have longer reaction times, that is, reaction times below their optimal level of functioning due to overarousal while extroverts were expected to optimize their performance on the task giving shorter reaction times.

It was intended that the research would identify any differences in physiological arousal between introverts and extroverts at identical caffeine doses, mirrored in differences in systolic and diastolic blood pressure and pulse between the two personality groups.

The second experiment followed directly using optimal reaction times and caffeine doses which were established in experiment one. It represented a practical application developed using the information already obtained in order to test the ability of subjects to generalize responses to a symbol which conveyed meaning. This experiment simulated a "real life situation" demanding a reaction time response within the confines of the laboratory setting. The assumption was made that subject responses in experiment two would closely mirror those of experiment one, thus supporting Eysenck's Personality Theory of Arousal based on the Yerkes-Dodson Theory (1908).

CHAPTER II

REVIEW OF THE LITERATURE

1. NEUROCHEMICAL AND ELECTROPHYSIOLOGICAL EVIDENCE OF CAFFEINE'S ACTION

Caffeine is one of the most widely used psychoactive drugs in the world. This is largely because it is socially accepted in most societies. It should also be borne in mind that many of the effects, both behavioural and cognitive exerted by this psychoactive drug are directly dependent on the dose at which it is administered.

Caffeine acts as a stimulant in the central nervous system at low to moderate levels whereas at extremely high levels, convulsions and death may ensue. However, for this to occur, consumption must be equivalent to 100 cups of coffee. Needless to say, this level is seldom reached. There are two mechanisms of xanthine action proposed to explain the effects of this widely used stimulant. The first is

(a) Phosphodiesterase Inhibition

On discovery of another enzyme (Sutherland et al., 1962) called phosphodiesterase, a second mechanism of xanthine action had been earlier proposed. This occurred after cyclic AMP was isolated and phosphodiesterase was found to be a degrading enzyme. Sutherland, Carr and

Mackintosh (1962) showed that xanthines could be used to inhibit phosphodiesterase and consequently increase the concentrations of AMP. This is important because cyclic AMP is known to be involved in synaptic transmission for several neurotransmitters, therefore showing caffeine to have stimulating properties in this process (Snyder & Sklar, 1984). Unfortunately, although this mechanism has a sound theoretical basis, further work by Snyder and Sklar (1984) has shown that substances far in excess of the moderate concentrations of caffeine ingested by individuals are required. Consequently, with the evidence to date, it appears that the stimulant properties of caffeine are more likely to be directed towards the adenosine receptors.

(b) Adenosine Receptors

According to Marangos, Boulenger and Patel (1984), caffeine has the ability to inhibit binding to two receptors in the brain, namely the benzodiazepine and the adenosine receptors. Caffeine is believed to inhibit binding to adenosine sites at a rate of 20 times that occurring at the benzodiazepine site. This implies that it is only at excessively high doses of caffeine that benzodiazepine receptors would be rendered inactive, while adenosine receptors may become inhibited at significantly lower caffeine concentrations (Marangos et al., 1984; Snyder & Sklar, 1984).

The brain attempts to compensate for the inactivity

of its adenosine receptors by receptor induction. This phenomenon is illustrated by research carried out by Marangos et al. (1984) who administered high doses of caffeine to white mice (400 mg/kg of diet to a 15-20 gm mouse) over a 16 and 23 day treatment period. The researchers estimated the mice would consume 2-3 grams of food each day, consequently the dose of caffeine administered would vary between 40-60 mg/kg/day. According to Gilbert (1981), this dose is representative of approximately 4-5 cups of coffee for humans when corrections are made for the weight of food consumed and the weight of the person. Hence, this dose was seen as appropriate because it represents an average level of consumption which can by no means be considered excessive.

At the 16 day inte val, there were significant increases in the number of adenosine receptors in the cerebellum and the brainstem. This increase in adenosine receptors was noted at the 23 day interval also and had also spread to other parts of the brain, however, the cerebellum and the brainstem remained the most significantly affected regions.

This paper lends support to those previous studies by Boulenger, Patel, Post, Parma and Marangos (1983) and Murray (1982). With respect to human behaviour, it has been suggested by Charney et al. (1984) that due to adenosine's inhibitory action on neurotransmitters and caffeine's ability to antagonize the adenosine receptors, increases in stimulation resulting from this mechanism

could well be displayed in overt behaviour and reflected in physiological parameters such as blood pressure and pulse.

Boulenger et al. (1983) summarized the two neuro-physiological effects of caffeine. Caffeine ingestion causes:

(1) blocking of adenosine receptors;

(2) chronic administration of caffeine elevates the number of adenosine receptors.

The elevation in the number of adenosine receptors is not to date fully understood. It is suggested that either,

(1) there is an increase in enzyme-adenosine uptake at the adenosine receptor sites, or

(2) the ligands (co-enzymes) and enzymes may be binding to different classes of adenosine receptors or both of these processes may be occurring.

Therefore, by suddenly withdrawing caffeine from an individual, the "enhanced adenosine mediated" effects are still evident and the amount of stimulation or depression which remains are recognized as withdrawal symptoms (Green & Stiles, 1986). This paper lends considerable support to the work of Snyder and Sklar (1984) and increased evidence of the subsequent effects on AMP in various tissues (Daly, Burns & Snyder, 1981; Sattin & Rall, 1970).

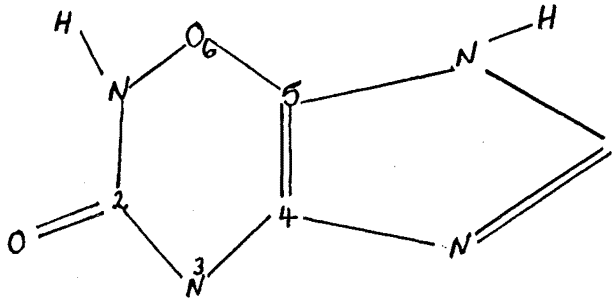
According to Freedman, Kaplan and Saddock (1975, 1978, 1981) the cortex is most sensitive to caffeine and changes in excitation such as increases in mental

alertness, more rapid, clearer thinking, restlessness and the ability to sustain prolonged intellectual input, can be brought about at quantities of as little as 100-200 mg of caffeine. The second most sensitive region of the brain to caffeine is the brainstem and thirdly the spinal cord, which requires extremely high concentrations of caffeine to become stimulated (between 2 and 5 grams).

2. THE XANTHINES

The xanthines comprise three very similar alkaloids, similar in chemical composition and properties. They occur in plants worldwide and are widely consumed among varied populations. For example, tea contains all three of the methylxanthines (caffeine, theophylline and theobromine) and is consumed by at least half of the world's population (Rall, 1980). Coffee occupies an important position in diet and it contains caffeine from Caffea arabica. Some carbonated beverages have caffeine in them, for example coca-cola contains naturally occurring caffeine and also caffeine added by the manufacturer (Graham, 1978).

Illustration 2. The basic xanthine structure



The structure of the three naturally occurring xanthines are (Rall, 1980):

Illustration 3. Caffeine

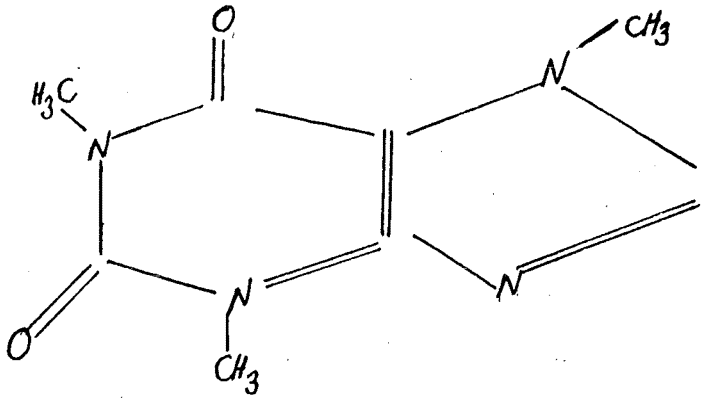


Illustration 4. Theophylline

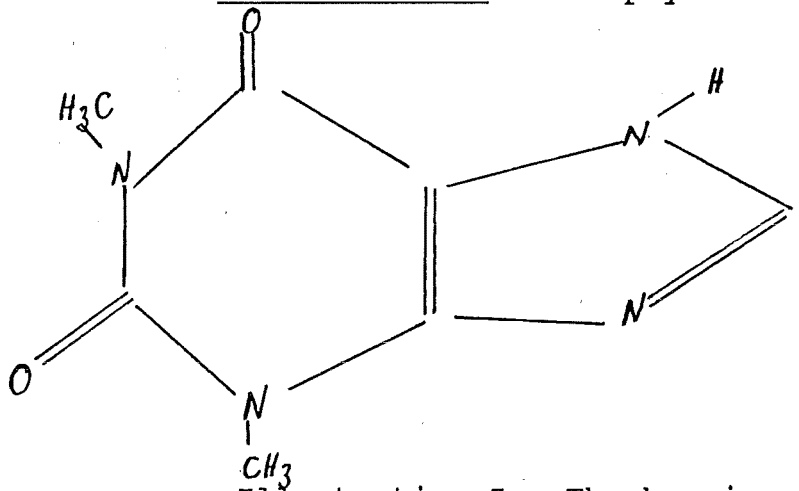
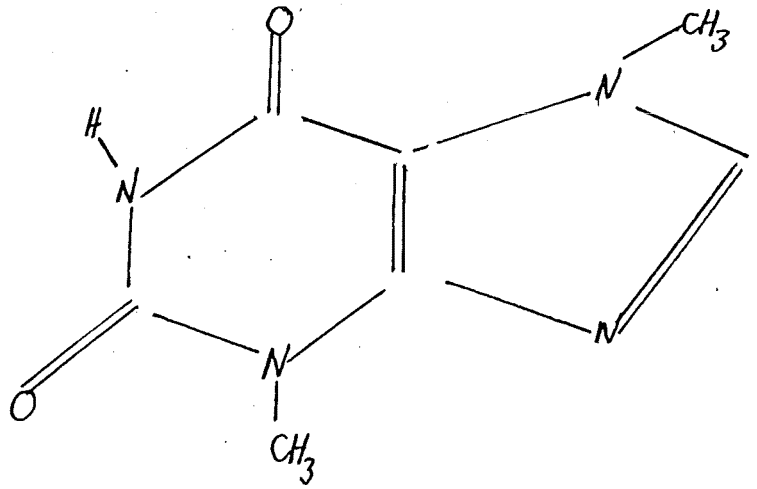


Illustration 5. Theobromine



If these substances are ingested in moderate amounts, they are known to produce therapeutic benefits, however when they are consumed at levels representative of overindulgence, they can produce poisoning reactions and they have been known to cause fatality in a few documented cases. As has been stated by Rall (1980), although the three naturally occuring methylxanthines share common properties, one may have greater therapeutic potency than the others in specific applications. Hence not all are used in relevant medications. Fatalities are rare, especially due to caffeine because at excessively high doses, caffeine is known to set up strong gastric irritation which usually causes vomiting before death can ensue. Alstott, Miller and Farney (1973) document the case of a female in her late 20's who overdosed on caffeine-containing pills. As stated by Rall (1980), solubility of the xanthines is very low and hence combination with other substances to form salts enhances the solubility of the xanthine. This means that once the complex has been absorbed into the body fluids, the substance breaks into the parent xanthine and related substance. Consequently, Alstott et al. (1973) were able to attribute death to the excessively high level of xanthine. According to Axelrod and Reichenthal (1954), caffeine is readily circulated throughout the body and easily absorbed. They showed that by taking the smallest concentration of caffeine in a tissue, the quantity ingested resulting in death could be estimated. This technique was implemented in Alstott et al's (1973) .

research and it was determined that 6.5 gm of caffeine had been ingested through the pills. This estimate supports Ritchie's (1970) estimation of between 5 and 10 grams of caffeine being representative of a lethal dose.

However, at reduced doses, the xanthines are known to produce therapeutic effects. They have a stimulatory effect on the central nervous system, a diuretic effect with respect to kidneys, a relaxing effect on bronchial muscle and stimulatory effects on cardiac muscle. These effects can be viewed as both therapeutic and detrimental, depending on the dose administered. For example, there is much evidence to suggest that xanthines increase the work that the heart must perform (Rall, 1980), while it has also been documented that xanthines can enhance coronary blood flow by dilating the arteries (Rall, 1980). These two conflicting views epitomize much of the literature surrounding the therapeutic use of xanthines and it still remains questionable as to whether the benefits offered to various tissues (such as the heart) by the use of the methylxanthines, outweigh the costs to these tissues in terms of extra work. As shown by Ogilvie, Fernandez and Winsberg (1977), between concentrations of 10 to 20 mg/ml, theophylline increases the heart rate while low concentrations of caffeine have been shown to slightly decrease the heart rate (Starr, Gamble, Marangolies, Donald, Joseph & Eagle, 1937). Consequently, concentration of methylxanthine plays a central role in determining the direction and extent of change in the tissue.

Research by Grollman (1930) suggested that small amounts of caffeine (0.16 gram) do not appear to affect the cardiovascular system while at doses between 0.30 grams and 1.0 grams, certain cardiovascular changes occurred, namely oxygen consumption increased by 36% at a dosage of 0.32 grams. He also observed an increase in the arterio-venous difference, which when examined in the light of the increased oxygen consumption, implies that the heart was required to work harder as a consequence of the caffeine.

At high concentrations of theophylline and caffeine or with excessive intake of caffeine-containing beverages, stimulation can cause tachycardia in some individuals which may culminate in arrhythmia. However, one of the most important situations for using caffeine and theophylline on the cardiovascular system has proven to be in the application of those xanthines to relieve congestive heart failure in emergency treatment situations. Piafsky, Sitar, Rango and Ogilvie (1977) suggested that xanthine involvement was important in developing more satisfactory methods of vasoditation because due to a lack of knowledge regarding the individual's sensitivity to the stimulating properties of xanthines, serious toxicity often ensued which placed the individual at serious risk. Related to the CNS, it is also thought that an association exists between coffee ingestion and increased serum cholesterol levels (Thelle, Arnesen & Forde, 1980). This, however, does not imply a causal relationship between caffeine

ingestion and serum cholesterol. Documentation of this relationship is, as with much of the literature, inconsistent and limited in its predictive value because no account was taken of body weight in the study.

Although it is extremely rare that death ensues as a consequence of caffeine toxicology (Diamais & Garriott, 1974) vomiting and convulsions are much more prominent. Insomnia, wakefulness and alertness are all symptoms of early toxicity and if caffeine concentrations are increased, they may lead to delirium and sensory disturbances. Respiration rate increases and the individual becomes tense and anxious (Rall, 1980). It is from this point that tachycardia and arrhythmia may occur.

(a) Diuretic Uses

According to Maren (1961), the methylxanthines, especially theophylline, increase the production of urine. This is of great therapeutic benefit to those individuals suffering from retention. This occurs by the xanthine exerting inhibitory forces on the reabsorption of sodium but there is no data to date depicting how successful the xanthines are in this respect.

Consequently, there is limited use of the methylxanthines in diuretic preparations for sale in the United States (Rall, 1980) and then only in subtherapeutic doses. This is because of the generalized stimulating properties of the methylxanthines. There is debate concerning the potency of both caffeine and theophylline but it is

recognized that at higher doses, side-effects such as nervousness, restlessness, tremor and insomnia can result. Nausea and vomiting have been shown to be common for theophylline at the upper end of the recommended therapeutic concentration (Jacobs, Senior & Kessler, 1976; Kordash, Van Dellen & McCall, 1977) and consequently because of the chemical similarity between the two methylxanthines there is reluctance to use caffeine in diuretic preparations in anything larger than subtherapeutic levels.

Another reason why caffeine is not favoured for its diuretic action is because of individual sensitivities. Some individuals may have the propensity to consume large amounts of caffeine with no adverse effects while another person consuming the same amount may suffer chronic poisoning as a result (Rall, 1980).

(b) Smooth Muscle Relaxants

The xanthines are recognized as successful smooth muscle relaxants when given orally and special attention is brought to bear on bronchial muscle, consequently the xanthines play a central role in the treatment of asthma (Rall, 1980). Caffeine was initially used to relax constricted muscles. However, theophylline has been shown to produce more beneficial effects than caffeine (Gong, Simmons, Tashkin, Hui & Lee, 1986). Consequently it is now the xanthine of choice to increase the capacity of smooth muscle. There is, however, a problem in the use of theophylline in the treatment of asthma, namely that both

theophylline and caffeine can elicit convulsions (Snyder & Sklar, 1984). Thus, therapeutic theophylline doses are approximately 10 mg/ml because convulsions have been observed at doses as small as 25 mg/ml. This poses serious problems as to the effectiveness of theophylline as a therapeutic intervention in the treatment of asthma because minimum adult doses are approximately 250 mg and as much as 750 mg may be required each day (Snyder & Sklar, 1984). This means that there is an ever-present risk of convulsion which limits the usefulness of the xanthines, so long as the stimulant properties of the xanthines are present. Theophylline is the xanthine of choice in the management of bronchiodilation as it is forty percent more effective at the same dose level as caffeine (Gong et al., 1986). This implies that caffeine may be used as the therapeutic intervention for mild obstructions, but even then, large quantities may have to be administered.

(c) Other Uses

According to Rall (1980) caffeine is often used in combination with other substances for the purpose of pain relief. Caffeine is used in combination with analgesics, especially asprin, in the treatment of ordinary headaches and it is also used with ergot as a treatment for migraine. It is believed that the caffeine has the ability to constrict the cerebral blood vessels which reduces tension and facilitates the action of the asprin or the

ergot respectively (Mathew, Williams & Wilson, 1985). Mathew & Wilson (1985) showed that Cerebral Blood Flow could be reduced by 250 mg caffeine for up to 90 minutes. However, this was not shown to be the case in a paper by Tokola (1985). He showed that while giving Tolfenamic acid (which is an anti-inflammatory analgesic) to 10 female patients, pain relief over a 5-hour period was not influenced by caffeine, at a dosage of 100 to 200 mg and caffeine did not facilitate the absorption of Tolfenamic acid. This study demonstrated that caffeine, in combination, may be of limited or no therapeutic benefit in the treatment of migraine when the participant exhibits no hypersensitivity to caffeine. However, this differs in the case of caffeine withdrawal headache. According to Dreisback and Pfeiffer (1943) and Greden et al. (1980), the caffeine withdrawal headache follows a specific developmental pattern. Both studies confir that "feelings of cerebral fullness" began which was followed by a painful throbbing throbbing headache, peaking 3-6 hours after the onset of the headache which was central in origin and becoming generalized and often accompanied by nausea and vomiting. It was discovered that if given early enough, caffeine provided the most effective treatment for this form of headache.

The Dreisback and Pfeiffer (1943) study depicted two patients who had migraines at the time the stimulant action of the administered caffeine was highest. They offered the following explanation which may in part

explain the findings of Tokola's (1985) study. They suggested that true migraines tend to occur as a result of caffeine overstimulation while caffeine withdrawal headaches occur when there is a deficit of caffeine. Although Tokola (1985) withheld fluids and foods from his participants for three hours, it has been stated by Rall (1980) that caffeine has a half life of 3.5 hours, consequently it may have been manifesting its effects in conjunction with the additional caffeine which was administered as part of the research. Consequently, care should be exercised in prescribing caffeine-containing substances for the relief of headache pain because caffeine withdrawal headaches may develop into caffeine dependency, and facilitate the development of migraines (Sawyer et al., 1982).

Caffeine has also been used as a central nervous system stimulant in the treatment of overdose by central nervous system depressants, such as opioids and barbituates. Caffeine was given by intramuscular injections, usually in combination with sodium benzoate (Rall, 1980). This method is no longer used because of ambiguity regarding amount to use and because of the ease and efficiency of more modern interventions.

Various other studies have been carried out regarding caffeine as a causative factor and its use in the treatment of a multiplicity of disorders of both psychological and physical origins. Included in this research are numerous studies on hyperactivity and attentional

deficit disorder (Dalby, 1985; Firestone, Poitras-Wright & Douglas, 1978; Garfinkel, Webster & Solman, 1981; Gilliland & Andress, 1981; Harvey & Marsh, 1978; Rall, 1980; Reichard & Elder, 1977; Schnackenberg, 1973).

Although the literature in this area is extensive, the methodological inconsistencies involving assessment techniques, particularly from study to study, makes comparison of findings virtually impossible. This aspect, coupled with limited information regarding the caffeine metabolism in children, makes any predictions on caffeine's role only tentative at this stage. The same may be said with respect to purely physiological disorders. This is perhaps most obvious in the literature surrounding caffeine and its relationship with cancer. Much of the literature is suggestive of causality between caffeine and various cancers (Bross & Tidings, 1973; Cole, 1971, 1973; MacMahan, Yen, Trichopoulos, Warren & Nardi, 1981; Stocks, 1970; Timson, 1977) while some is suggestive of a reversed relationship (Posner et al., 1986) and others are noncommittal and realize the need for further research (Posnick, 1985). On the basis of the information from these studies, restraint may be advocated. However, because of the narrow focus commanded by much of the literature, there may be many more factors involved in association with caffeine which bring about various forms of cancer. Because of the lack of information regarding such factors as subject representativeness, subject sensitivity, consumption level and duration with respect to body weight,

any clinical significance that these studies might have is questionable.

3. THE RELATIONSHIPS BETWEEN CAFFEINE AND REACTION TIME

It has previously been widely documented that reaction time is a useful parameter in the investigation of the stimulatory properties of caffeine (Hollingsworth, 1912). Reaction time is also used in the evaluation of individuals with cerebral dysfunction, either pharmacological, psychotic or medical in nature where it is impossible to administer test batteries (Elsass, 1986). Elsass has shown by administering a reaction-time task to a heterogeneous group of individuals, that cerebral dysfunction could be detected, and more accurate detection of progressive diseases result. This can only occur, according to Elsass (1986) when the individual has a definable disorder which lessens the usefulness of the technique. However, it provides a technique which has the potential to be exceedingly useful in clinical settings when more is understood regarding neurological processing during reaction-time tasks.

According to Rall (1980), one of the side-effects of caffeine ingestion is an ability to move rapidly and clearly process thoughts and a decrease in reaction time. According to Goldstein, Kaiser and Warren (1965), typists work at faster rates with fewer errors as a result of

caffeine consumption. However, this may not be the case in tasks involving delicate muscular movements (Carpenter, 1962). Rall (1980) suggests that, after allowing for individual differences, between 85 and 250 mg of caffeine is all which is required for reaction time to decrease. This is supported by the research of Goodman and Gilman (1980), Nash (1962) and Osborne and Rogers (1985), who suggest that reaction time is reduced at doses of between 80-200 mg of caffeine. The doses of caffeine vary slightly between studies and evidence regarding the effects of caffeine on various tasks is often conflicting. For example, Schilling (1921), showed how caffeine retarded reaction time. However, there were shortcomings in this research, such as no dose specificity was given for individuals. It is highly unlikely that a uniform dose of caffeine would have the same effects on all individuals. This could provide one reason why there was such diversity in the physiological measures obtained. It is questionable that the initial dose of 5 grains of caffeine is an ambiguous measurement and also that if this measurement had been more accurate, and perhaps varied, the results could have been more meaningful.

An individual's body weight and recent food consumption are also two variables which affect response rates and consequently both of these variables will affect each individual's rate of absorption of the caffeine. Also, as Schilling (1921) points out, fatigue must play a central role in increasing reaction time as the session

gets longer. Contrary to the research of Schilling (1921), Horst and Jenkins (1934) and Horst, Robinson, Jenkins and Bao (1934) researched the effects of caffeine and decaffeinated coffee on blood pressure and pulse rate with respect to reaction times and specific motor reactions in both young men and men of various ages.

Horst and Jenkins (1934) made the observation that average pulse rates decreased after caffeine was administered to a group of young men, to become lower than the pulse rates of those who had consumed decaffeinated coffee. However, it is not easy to generalize from these findings because they also found that often the pulse rates from those consuming caffeine were very similar to or even faster than those who were not. Pulse rates have been shown to vary in this experiment and conclusions as to why this occurred can not be directly attributed to any specific variable. Horst and Jenkins (1934), also showed a decrease in reaction times among some of the younger and older men at doses as small as 2 mgm of caffeine/kg body weight (however, some individuals manifested longer reaction times at 2 mgm caffeine/kg body weight).

The average reaction times of the older participants were characterized by greater variability than for the younger men. However, over the course of the research, this did not generally surpass the variability of the participants in the no caffeine condition. It was frequently found, however, that those individuals consuming caffeine exhibited longer reaction times on the

following day of experimentation and that approximately two hours after caffeine consumption, blood pressure tended to increase beyond blood pressure after decaffeinated consumption. This was the case with some of the younger men but more especially among the older ones (Horst & Jenkins, 1934). The changes demonstrated by these two researchers show that there is much variance in both pulse and blood pressure while reaction time tends to be faster at caffeine doses of 3, 4 and 4.5 mgms of caffeine which are comparable to the doses used in this research. (One point which is common to this paper and also to that of Schilling (1921) and may pose as a limiting factor is that in neither experiment were adequate initial measures obtained of pulse, blood pressure and reaction time. That is, no measurements were carried out before caffeine was administered. This raises questions as to how representative their data may be and it could partly explain the degree of variability in the results of the two experiments.) This characteristic is also present in the research of Horst et al. (1934) in their examination of the parameters with respect to certain motor reactions. A second problem of subject anticipation becomes apparent in this paper. The caffeine was administered in capsules in 300-400 cc of boiling water, while on caffeine-free days, milk and sugar replaced the caffeine. It is unclear as to how this was done or if subjects were aware of the difference. Consequently, it is likely that subjects' anticipatory effects may affect the subjects'.

responses. Although they showed caffeine to have detrimental effects on acquired motor skills, it would have been more appropriate to employ an alternative methodology, incorporating both pre and post measurement of the physiological parameters.

Research has also been carried out by Osborne and Rogers (1983) on the relationship between alcohol and caffeine and its effects on reaction times. This paper helps to dispel the widely-held view that caffeine has an antagonistic effect on al ɔhol and therefore reduces the effects of alcoholic beverages. Osborne and Rogers (1983) used a comprehensive methodology involving eight subjects who served as their own control in four experimental conditions:

1. No alcohol and no caffeine.
2. No alcohol and caffeine.
3. Alcohol and no caffeine.
4. Alcohol and caffeine.

They showed that caffeine served to enhance the detrimental effects of alcohol. This finding is contrary to expectations in view of the fact that one is a central nervous system stimulant nd the other a depressant. Pilcher (1912) suggested that the pharmacological specificity of each compound may not be as straightforward and there could well be both stimulating and depressant elements to each compound. Also, as Osborne and Rogers (1985) point out, differences in reaction times may occur at various dose combinations of the alcohol and caffeine.

A further factor which may have contributed is that of differing subject sensitivity. Nash (1966) expands on this by suggesting that any research into the joint action of drugs will face limitation and problems of limited dose levels and limited numbers of subjects. It is exceedingly difficult to predict the outcomes of these studies, as task demands vary so much from study to study (Nash, 1966).

As was stated by Weiss and Laites (1962), inconsistencies emerging in the data with respect to reaction time must, at least in part, be attributed to methodological differences. An example of this precise point is demonstrated by comparing the research of Cheney (1935) and Horst and Jenkins (1934). Both papers have focused on the direction and magnitude of the change in reaction time brought about by caffeine. They differ in that Horst and Jenkins (1934) administered doses between 3-4 mgm/kg body weight while Cheney 1935) set an upper limit of 5.6 mgm/kg of body weight. However, all of the researchers agree that reaction time was decreased approximately two hours after the task. Unlike Horst and Jenkins (1934), Cheney (1935) did not find any detrimental effects on reaction time the following day. This could be because, although the two experiments were well designed and incorporated strong methodologies, they were measuring two completely different things. Cheney (1935) was measuring stimulus discrimination which can not be considered as simple reaction time because the subject was required to

employ cognitive processes to determine the appropriate response to make to the light stimulus. Therefore, decisions had to be made by the participants of Cheney's (1935) experiment while no decision-making process was employed by participants of Horst and Jenkins' (1934) research. Consequently, meaningful comparisons cannot be made between the two papers.

4. THE EFFECTS OF CAFFEINE ON MOOD

Caffeinism develops among individuals who ingest caffeine at significantly high levels and they develop dependency. Greden (1974) suggests that this can occur after a duration of habitual use of 500 mgms of caffeine per day. This would tend to suggest that many individuals have caffeinism to some degree. As with any substance dependency, the symptoms manifest themselves after a period of withdrawal, which is usually only a matter of two to three hours in the case of caffeine. The most common characteristics are, as have been previously documented, tremor, irritability, tense muscles, insomnia, wakefulness, headache and even heart palpitations and arrhythmia. These are all mood-related symptoms and consequently it has been widely documented that associations exist between various emotions and caffeine consumption. As Greden (1974) has pointed out in three case reports, high intake of caffeine can promote symptoms indistinguishable from those of anxiety neurosis. These

symptoms have also been seen to occur when individuals attempt to reduce caffeine intake, and have consequently become recognized as withdrawal symptoms which usually culminate in caffeine withdrawal headache.

Much research has been carried out to investigate the nature of relationships between caffeine and various emotions (Charney et al., 1984; Charney, Heninger & Jatlow, 1985; Cherek, Steinberg & Brauchi, 1984; Downing & Rickels, 1981; Greden, 1974; Greden, 1985; Greden, Fontaine, Lubetsky & Chamberlin, 1978; Lee, Cameron, & Greden, 1985; Lutz, 1978; Sawyer, Julia & Turin, 1982; Svensson, Persson & Sjoberg, 1980; Winstead, 1976). However, anxiety remains the most widely investigated emotion with respect to caffeine consumption and these investigations have yielded some startling results.

Anxiety has been shown to become manifested in various physiological areas, perhaps one of the most unpleasant of these being documented by Lutz (1978) as the restless legs syndrome. This creeping sensation occurs in connection with caffeinism as do many other characteristics such as insomnia, heightened awareness and irritability. Lutz (1978) proposed that caffeine is a major proponent in the development of the restless legs syndrome, which can range from mild to severe. Depression has been seen to develop, secondary to anxiety, and this may be in part a consequence of reduced sleep brought about by the syndrome. However, it has not as yet been directly associated with chronic caffeine ingestion, or what has come to

be recognized as caffeinism.

An interesting feature in the literature surrounding caffeine ingestion and anxiety is that proposed by Lee at al. (1985). They conducted research involving anxiety disordered patients and hospitalized medical patients. They suggested that rather than anxiety levels being inflated among heavy caffeine consumers, the anxiety levels increased among those individuals who appeared in the Hopkins Symptom Checklist to have a heightened level of sensitivity towards caffeine. This lends an interesting twist to the literature regarding caffeine and anxiety and it implies that as well as physiological withdrawal, heightened subject sensivitity may also be central to increasing anxiety. The next step in the research could be to determine those variables which facilitate heightened sensitivity at both a physical and psychological level.

Several research papers have been carried out to investigate the effects of caffeine on individuals who are already anxiety-disordered. Contrary to expectation, as Myung, Cameron and Greden (1985) point out, instead of becoming more greatly anxiety-disordered, these individuals appear to have a heightened caffeine sensitivity, as was previously depicted by Lee at al. (1985) and they refrain from high caffeine consumption. Freitas and Schwartz (1979) conducted some research with chronic psychiatric patients in order to determine whether or not any improvements in behaviour resulted from reducing or

eliminating caffeine. The results state that when decaffeinated coffee was administered, hostility and anxiety decreased. However, this research manifests several methodological weaknesses. The patients were given "limited access" to a coffee dispensing machine over the duration of the research. This is grossly ambiguous as no exact information exists regarding amount of caffeine consumed. It is highly likely that most of the participants were receiving medication also. However, no mention of this is made and one can but assume this point was overlooked. Charney et al. (1985) lend support to the research of Myung et al. (1985) in their study which investigated anxiety effects of caffeine in panic-disordered individuals. They proposed that because caffeine is recognized as an adenosine receptor agonist, panic and anxiety disordered individuals may have abnormal neuronal systems with respect to adenosine, resulting in heightened sensitivity. It is believed that such neuronal effects of caffeine ingestion may well serve as a partial reason for symptoms of depression. Greden et al. (1978) showed a combination of both anxiety and depression symptoms among a sample population of 84 hospitalized psychiatric patients, such as fatigue, crying, and worrying. It has been suggested by Soloman and Corbit (1974) that the depressed state may be set up in response to the caffeine induced heightened anxiety state by some form of biological automation. This being the case, successive attempts to determine the etiology of depression may

become confused.

Aggressive behaviour is the third major emotion reportedly influenced by caffeine ingestion. The major contributions in this area have been made by Cherek (1984) and the literature is not vast. Cherek et al. (1984) employed a new methodology which involved evoking aggressive responses from subjects by making them forfeit a sum of money to a fictitious person. This procedure was carried out under three treatment conditions, each of which lasted 30 minutes.

(1) Decaffeinated coffee - 4 teaspoons.

(2) Regular and decaffeinated coffee - 2 teaspoons of each.

(3) Regular coffee - 4 teaspoons.

They showed that when subjects were in treatment conditions (2) and (3), the amount of aggressive responding decreased. This change was not mirrored in the decaffeinated coffee condition. These results are similar to those obtained by Cherek, Steinberg and Brauchi (1983) with respect to caffeine. Therefore, the decreases in aggressive responses could be attributed to caffeine and no other constituent of coffee. Another interesting point was that those subjects who normally drank larger amounts of coffee showed a greater suppression in aggressive responding than those whose normal consumption was lower. Thus subject sensitivity played a central role. It is unfortunate that no physiological parameters were employed in this research, such as blood pressure and pulse.

Consequently, it is not possible to determine whether suppressed aggression is directly attributed to caffeine, or as is suggested in the case of depression, the result of a biologically automated process (Soloman & Corbit, 1974).

5. THE EFFECTS OF CAFFEINE ON PERSONALITY

Much research has been carried out regarding the interplay between various personality attributes and caffeine ingestion, with emphasis on introversion/extroversion. One of the most widely used personality scales is the Eysenck Personality Inventory. However, controversy still reigns as to the predictive qualities of the introversion/extroversion dimension compared with the subscales of sociability and impulsivity. Carrigan (1960), Guilford (1975, 1977), Revelle, Amard and Turriff (1976) suggested that these subscales functioned as more accurate parameters of performance on a task than the introversion/extroversion scale itself. Not surprisingly, Eysenck and Folkard (1980) rebutted this view. Regardless of the stance adopted by researchers, personality characteristics have been linked with caffeine effects on specific tasks. Fundamental to any research in this area is the assumption of Eysenck (1963) that the typical arousal levels of individuals as measured using the Eysenck Personality Inventory with the introversion/extroversion dimension, are due to differences in physiological arousal

in response to environmental stimuli of identical strengths. He suggested that introverts demonstrate higher arousal than do extroverts at the same stimulus strengths (Erikson, Hager, Houseworth, Dugan, Petros & Beckwith, 1985). By adopting Eysenck's (1967) assumption, the administration of caffeine should enhance the differences between the two personality types. This is certainly the case as reported by Gilliland (1980) who showed that at low levels of caffeine, introverts were found to increase in speed and accuracy on a verbal performance task. Extroverts demonstrated decrements in performance at low levels of stimulation which significantly increased at higher doses of caffeine. Bowyer, Humphreys and Revelle (1983), showed, using the subscales of impulsivitity (from the Eysenck Personality Inventory) that high impulsives when given placebo declined in their recognition of word lists while low impulsives showed a smaller decline. Caffeine significantly improved recognition memory according to Bowyer et al. (1983) which lends support to the Eysenck theory of differing physiological arousal levels among personality types. Regardless of the debate with respect to the accuracy of specific subscales reported in the literature, research indicates that caffeine improves the performance of extroverts on simple tasks such as vigilance tasks (Revelle, Amaral & Turriff, 1976) and impairs that of introverts. However, the findings of these studies are dependent on doses administered and, to a large extent, on the methodology employed within

specific studies.

While some proponents of Eysenck's theory suggest greater relevance from using the impulse subscale of the inventory, Anderson and Revelle (1982), Eysenck and Levy (1972), Eysenck and Follard (1963) and Revelle at al. (1976) and the subscales of impulsivity and sociability, Guilford (1975, 1977) and Carrrigan (1960), Eysenck (1977) strongly refutes this position. Despite whatever shortcomings Eysenck's theory may have, it provides a useful framework in which to investigate arousal and by adopting the attitude of Bowyer, Humphreys and Revelle (1983), it is only by carrying out many studies and involving many variables that insights into arousal will be forthcoming. Thus, although the Yerkes-Dodson (1908) Curvulinear Theory of Arousal is largely descriptive, it provides a basis on which to evaluate the merits of successive theories and it also provides a useful framework into which variables can be incorporated to be systematically investigated with respect to their association with personality.

6. TOLERANCE TO CAFFEINE

Limited information is available regarding the development of tolerance to the stimulating properties of caffeine in humans, probably because when compared with the actions of many other drugs, the actions of caffeine are quite minor (Chou, Khan, Forde & Hirst, 1985). Most of the research in this area has been conducted using lab-

oratory rats. Marangos, Boulenger and Patel (1984) suggest the most logical site of action being that of the adenosine receptors. They demonstrated the development of adenosine receptors earlier in mice pups who had received chronic amounts of caffeine while still in utero. They also demonstrated that once the mice pups reached adulthood, they possessed a larger number of adenosine receptors. Chou et al. (1985) demonstrated an increase in firing rate of reticular neurons in rats who had not previously received caffeine. This occurred at doses of 2.5 mg/kg. The suggestion is made by Chou et al. (1985) that due to the increased number of adenosine receptors, uptake of adenosine becomes more efficient. This is known as up-regulation and is thought to be the mechanism which underlies caffeine tolerance.

In the literature, tolerance to caffeine is demonstrated by fewer obvious effects among those individuals who are constant users when compared to abstainers who ingest a similar amount (Goldstein, Kaiser & Whitby, 1969). They also suggest that individuals have caffeine consumption levels due to an interplay between tolerance and specific sensitivity levels, unique to the individual (Hayre, 1973).

CHAPTER III

METHODS SECTION FOR EXPERIMENT ONE

(a) Subjects

Thirty-four subjects from the stage one Psychology course at the University of Canterbury participated in this research. Of the total number of thirty-four participants, fourteen were male with an age range of 18 to 23 years, and a mean age of 20.15 years. Twenty females participated with an age range of 17 to 35 years, and a mean age of 22.0 years.

Requests were made of the 157 students from the morning lecture group of the stage one Introduction to Psychology course to participate in some research into the effects of caffeine on Reaction Time. From this request, 87 students completed the Eysenck Personality Inventory and the caffeine consumption survey (see Appendix 1) and indicated their interest in taking part. The remaining 70 students either handed in incomplete or incorrect inventories and surveys, or indicated that they did not wish to take part in the study.

From the 87 completed inventories and caffeine surveys, the extremes on the Introversion Extraversion continuum were established at Introverts $\leq$ 10 and Extroverts $\geq$ 16. This meant that a large number of individuals were excluded from the study and the research population comprised 34 subjects, who showed strong characteristics

of their appropriate group. The two personality groups comprised both caffeine consumers and abstainers and also those who consumed caffeine but not each day. The 34 subjects were randomly assigned to either the placebo group which comprised 7 males and 9 females or an experimental group of 7 males and 11 females.

(b) Apparatus

The apparatus used was the same for each session and all research was conducted in the same location. Heart rate, systolic and diastolic blood pressures were recorded using an electronic Sphygmamanometer, model MI-100 which gave a digital readout of the three required variables in mm/Hg obtained through a standard adult size cuff. The MI-100 Sphygmamanometer was the most reliable and advanced pattern recognition of blood pressure available to provide accurate systolic and diastolic readings. All reaction time testing, for both experiments, was carried out using an Apple IIe keyboard and disc drive with a Mitsubishi Electric display monitor. The programmes for each of the two experiments were held on Maxell MD2-D standard 130 Kilobite floppy discs.

(c) Procedure for Experiment One

Experiment 1 consisted of three sessions for experimental subjects and one session for the placebo group. Thus the two groups were not directly comparable. Because of the disparity between the number of sessions, it was necessary to request subjects not to discuss the

research with others.

All subjects were blind as to which group they were a member of and they were only told that they would receive various amounts of caffeine and their reaction-times monitored. Attempts were made to conduct the experimental sessions on alternate mornings. However this was not always possible because of timetable restrictions on some of the participants. Orange cordial was selected as the medium in which the pure caffeine was mixed. The subjects of both the placebo and experimental groups received 20 ml orange concentrate dissolved in 130 ml water. The amount of caffeine each subject received was determined on the basis of the consumption survey, and consequently differed for each participant. This detailed dose appropriate method was used in an attempt to determine an average consumption level for each individual involved in the study. Previous research carried out by Rall (1980), placed the quantity of caffeine required to decrease reaction times at between 85-250 mgs. Similar doses have been determined by Goodman and Gilman (1980), Nash (1962), and Osborne and Rogers (1985).

By determining the amount of caffeine each subject received on the basis of their responses to the consumption survey, the intention was to ensure the most appropriate dose for each individual. This was done in an attempt to establish more specific information regarding the amount of caffeine required to decrease reaction time with respect to personality type while also taking the

individuals chronocity of use into account. The initial dosage of caffeine was determined using question 3 of the Caffeine Survey (see Appendix 1). If individuals indicated 1 cup consumed in the morning, then the equivalent of 1 cup of coffee was given (65 mg of caffeine). If the individual indicated 2 cups consumed in the morning, then the equivalent of 2 cups of coffee was administered (130 mg caffeine). This was the method used to establish the initial dose for each subject in the experimental group. Increments of 50 percent were made over the second and third treatment sessions which the subjects remained unaware of.

The initial dose level was determined by identifying the quantity of coffee consumed in the morning, rather than the quantity consumed over the course of the entire day. This was because several participants had high levels of consumption and it would have been ethically questionable as to whether these levels could be safely increased in increments of 50 percent. Also, because it was better to carry out testing before any coffee had been consumed, the majority of testing occurred in the mornings, consequently average doses were determined by taking these constraints into account.

The caffeine content figures per cup of coffee were taken from Gilliland and Bullock (1983-84) and although they may present conservative estimates of the quantities, when considering the estimates given in other research (Dalby, 1985; Greden, Victor, Fontaine & Lubetsky, 1980;

Sawyer, Julia & Turin, 1982; Srisuphan & Bracken, 1986) they were believed to be reasonably representative.

Each session commenced in the same room with the subject being presented a pre-test of visual analogue scale depicting six opposing moods. The instructions were standardized on the form (see Appendix 2) but were verbally elaborated on for those subjects requiring further identification. The six moods of relaxed, not breathless, sleepy, unhappy, calm, confidence-lacking and their opposites were selected because these anxiety-related moods have dominated the literature on caffeine and because some of the participants had reported similar subjective feelings in the caffeine survey.

Pre-test measures of systolic and diastolic blood pressure and pulse were taken for each session using the Sphgmamanometer described previously. This was immediately followed by the reaction-time task which required the subject to position him/herself at the computer and respond to the following instructions.

"Make yourself comfortable.

Hit any key when ready to continue.

Type your first name please and then hit the <return> key. The return key is the odd shaped key on the right side f the keyboard.

Thank you.

Shortly you will be shown a blank screen. It will stay blank for a few seconds and then suddenly a blob will appear near the middle.

When you see this happen, hit the space bar as quickly as possible.

The space bar is the long bar in the bottom row.

Try hitting it now.

SPOT ON.

Now let's get on with the test. There will be about 20 trials for you to react to. It shouldn't take very long if you concentrate.

Hit the space bar to begin."

The computer program used a rectangular shape which flashed onto the centre of the screen. Two randomized sequences were used to ensure that durations between presentations of the shape would be different in the pre and post measurements. This randomizing of durations eliminated practice effects by making it almost impossible to anticipate with any degree of accuracy, the duration which would elapse between the successive shape presentations. Once this task was completed, the mean reaction-time was recorded and the subject was told they had performed well. They were then given the orange cordial with appropriate amounts of caffeine. The second half of the session commenced 30 minutes after the caffeine was ingested because according to Greden (1979) and Marks and Kelly (1973) the effects of caffeine are maximized 30-45 minutes after ingestion. The second half of the session mirrored the first half exactly, and this procedure is standardized for all sessions (using different caffeine doses) and for each participant with the instructions

being given at the commencement of each computer trial.

CHAPTER IV

METHODS SECTION FOR EXPERIMENT TWO

(a) Subjects

The second analysis involved the cooperation of the 18 individuals who participated in analysis one. A further 16 individuals who comprised the control group also took part. The 18 individuals of the experimental group received the caffeine dose in the second analysis which yielded the best mean reaction time in analysis one, while the control group received no caffeine. The assumption was made that the rapid reaction time of analysis one at the specific dosage, would be mirrored when the rectangle was replaced with a road sign at the same caffeine dose.

(b) Apparatus

The apparatus and location for experiment 2 were an exact replication of those used in experiment 1. The caffeine, orange drink and all dependent variables were measured in exactly the same way as for experiment 1.

(c) Procedure

Experiment 2 consisted of one session for each subject of both the experimental and placebo groups, who received orange cordial with no caffeine. The session commenced by giving the subject the pre-test visual analogue scale with standardized instructions which were used in the previous experiment. Pre-test measures of

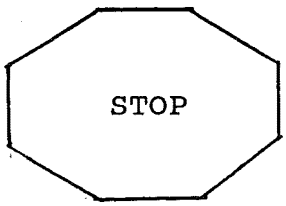
systolic and diastolic blood pressure and heart rate were also obtained. This was immediately followed by the pre-drug reaction-time task using the computer. The same instructions as used in experiment 1 came onto the screen and two verbal amendments were made to these.

(1) The subjects were told that a roadsign, rather than a blob would appear, and

(2) The computer only presented a trial of 10 signs, rather than the 20 of experiment 1. So as to standardize the number of roadsigns and rectangles that were presented, the roadsign program was carried out twice.

The roadsign used was a stop sign depicted below and it differed from experiment 1 in that it changed positions on the screen.

Illustration 6. The Stop Sign



A stop sign was selected because it was the fastest sign the computer could generate and consequently most appropriate for the nature of the research. After the pre-drug mean reaction-time was recorded, the subject was told he/she had performed well and the orange drink was

administered with appropriate amounts of caffeine dissolved for members of the experimental group. The second half of experiment 2 mirrors the first half and after the post-treatment measures on the dependent variables, the subject was again positively reinforced verbally and the session concluded.

(d) Summary of Steps in Each Experimental Session

(1) Pre-drug - Systolic Blood Pressure
Diastolic Blood Pressure
Heart rate.

(2) Pre-drug visual analogue of mood scale:

1. Relaxed/Restless
2. Not breathless/Breathless
3. Sleepy/Alert
4. Unhappy/happy
5. Calm/Nervous
6. Confidence lacking/Confident

(3) Pre-drug Reaction-time task.

(4) Post drug - Systolic Blood Pressure
Diastolic Blood Pressure
Heart Rate

(5) Post-Drug Mood-Scale

1. Relaxed/Restless
2. Not breathless/Breathless
3. Sleepy/Alert
4. Unhappy/happy
5. Calm/Nervous

6. Confidence lacking/Confident

6. Post-drug Reaction-Time

(e) Control Features

Attempts were made to ensure that the strictest controls possible were maintained so as to increase the reliability of the study and to enable its replication. Standardized instructions were given for each of the sessions of experiment 1 and instructions only differed between the two experiments as much as was necessary to ensure that subjects understood that the second computer program differed from the first.

Because of differences in student timetables, the ideal situation of conducting all sessions at the same time each day was not possible. However, it is believed that any bias from such a source would be minimized through the standardized nature of the instructions and feedback and because the total number of sessions for each participant occurred over the shortest time-span possible. Orange cordial was used to mask the bitterness of the added caffeine. This was essential in order to disguise the quantities being administered to subjects. They believed they were receiving caffeine, including members of the placebo group but they were blind as to how much. It was also important to request subjects not to consume tea or coffee for several hours before the session. It is assumed that subjects complied with this request. However, there is no evidence indicating that this occurred

100% of the time and there were no physiological measures to confirm this.

CHAPTER V

RESULTS OF ANALYSIS ONE

The data were analysed using BMDP2V software based on an analysis of variance on repeated measures for unequal cells design. This form of analysis was employed for both the first and second analyses. The independent variables for the first analysis were sex, sessions (B) and personality type (I) and there were 18 participants who constituted the experimental group. Each of the 18 participants underwent three sessions in which scores on the selected dependent variables (systolic and diastolic blood pressure, heart rate, reaction time and the six mood rating scales) were obtained prior to caffeine administration and after caffeine administration. The specific details of these sessions can be found in the procedure subsection of the methods. A table of the significant results for analysis one follows accompanied by a discussion of these.

TABLE 1 BMDP2V UNEQUAL CELLS ANALYSIS ONE

		F	TAIL PROBABILITY
1.	PRE-CAFFEINE SYSTOLIC BLOOD PRESSURE		
	SEX	12.15	0.0036
	SESSION	8.81	0.0011
	SESSION, PERSONALITY, SEX	3.40	0.0477
2.	PRE-CAFFEINE DIASTOLIC BLOOD PRESSURE		
	SESSION	6.65	0.0043
3.	PRE-CAFFEINE MEAN REACTION TIME		
	PERSONALITY	4.83	0.0453
	SESSION,PERSONALITY	7.25	0.0029
	SESSION,PERSONALITY,SEX	4.35	0.0227
4.	PRE-CAFFEINE MOOD 2 (SLEEPY/ALERT)		
	PERSONALITY,SEX	4.44	0.0535
5.	PRE-POST CAFFEINE SYSTOLIC BLOOD PRESSURE DIFFERENCE		
	SESSION	4.81	0.0161
6.	PRE-POST CAFFEINE DIASTOLIC BLOOD PRESSURE DIFFERENCE		
	SEX	6.36	0.0244
	SESSION	3.75	0.0361
7.	PRE-POST CAFFEINE MEAN REACTION TIME DIFFERENCE		
	SESSION,PERSONALITY	3.65	0.0391
8.	PRE-POST CAFFEINE MOOD 1 (RELAXED/RESTLESS) DIFFERENCE		
	PERSONALITY,SEX	4.59	0.0502
9.	PRE-POST CAFFEINE MOOD 2 (NORMAL BREATHING/BREATHLESS) DIFFERENCE		
	PERSONALITY,SEX	7.74	0.0147
	SESSION	5.01	0.0138

1. ANALYSIS ONE

1. Although significant sex and sessions effects occurred with pre-caffeine phase systolic blood pressure, they were more appropriately considered in the light of the significant sex x sessions x personality type inter-action. Figure (1) shows a systematic decrement over sessions for introvert females and extrovert males and, in general, males had higher systolic blood pressures than females.
2. Figure (2) demonstrates a significant effect with pre-caffeine phase diastolic blood pressure with respect to sessions. There is a systematic decrease in diastolic blood pressures over all sessions for each group, with the possible exception of the second session for introvert males which is slightly lower than the third session but not significantly so, since interactions were not significant.
3. There were no results of significance for the third physiological parameter, that of heart rate, as is demonstrated in figure (3).
4. Figure (4) demonstrates the pre-caffeine phase mean reaction time scores. These scores were obtained by averaging the 20 reaction-time tasks which were presented in each session. Although significant personality type effects occurred during this phase, they were more appropriately considered in

light of the significant personality type x sessions x sex inter-action. Figure (4) shows greater uniformity over sessions for females than males but no other obvious trend. There is also a significant interaction effect between sessions and personality type.

5. The mood statistics which were obtained from the visual analogue scales did not yield any significant results in the three pre-caffeine phases (see figs 5, and 7-10), except for a weak interaction effect between personality and sex with respect to the three measures of mood 2 (Not breathless/ breathless). See figure 6. The extrovert males and females demonstrate a higher score of breathlessness in the third session than either the first or second sessions while the reverse is observed for introvert males and females.

2. PRE-POST CAFFEINE DIFFERENCES FOR ANALYSIS ONE

The pre-post caffeine differences were obtained by expressing the post-caffeine measure as a fraction of the pre-caffeine measure, subtracted from 1 to yield a proportion. This method was selected because it did not require converting results into percentages. This was important because expression of mood differences as percentages of a visual analogue detracts from the meaningfulness of the instrument, especially when negative

results occurred. The negative scores in this section of the analysis show that the pre-post caffeine difference was in the negative direction while a positive score was in the positive direction. That is to say, that the post caffeine measure on the dependent variable was larger than the pre-caffeine measure. In conclusion, the pre-post caffeine sessions provide a discussion of the difference between difference scores, rather than specific scores.

1. Figure (11) demonstrates a significant sessions effect in the pre-post systolic blood pressure difference phase. Figure (11) depicts positive pre-post systolic blood pressure differences for all three groups except extrovert females on the initial session extrovert females on the initial session followed by a negative movement in the second session which tended to a positive difference in the third session of the pre-post systolic blood pressure difference phase. The introverts demonstrated this trend more strongly with the largest difference occurring between the first and second sessions.

2. In the pre-post diastolic blood pressure difference phase, sex had a significant effect on these differences. Males yielded larger positive diastolic blood pressure differences than females for all three sessions (see figure 12). There was also a significant sessions effect in which the positive difference of the initial session was followed by

decrements in difference in session two for each group except extrovert females where the differences were smaller anyway. These trends can be seen in figure (12).

3. In the pre-post mean reaction time difference phase, there was a significant sessions x personality interaction. Introverts demonstrated a trend of positive difference followed by two sessions of negative difference while, as figure (14) demonstrates, the extroverts showed the opposite trend of an initial and second session negative difference followed by a positive difference on the third session.

4. In the pre-post mood 1 (Relaxed/Restless) difference phase, there was a significant personality type x sex interaction (see figure 15). Introvert males showed stronger positive differences in all three sessions than introvert females who showed a reverse trend with all three sessions being positive. Extrovert males and females differences were not as large as the introverts and they tended to be negative for the first two sessions and positive for the third.

5. Figure (16) demonstrates the pre-post mood 2 (Not breathless/Breathless) differences. There was a significant sessions effect demonstrated via all first session differences being positive, followed by decrements in second session differences which

were then followed by increments in third session differences. This trend was demonstrated for all participating groups with respect to pre-post mood two differences and was more strongly evident among introverts when compared to extroverts. There was also a significant personality type x sex interaction. See figure (16).

CHAPTER IV

RESULTS ANALYSIS 2

The 18 participants from the first analysis returned for a further session during which the caffeine dose which yielded the best mean reaction time from analysis one was readministered. These results were compared with 16 individuals who received no caffeine and underwent similar testing.

The second analysis differed from the first in two respects. Firstly, the grouping variable, sex, was excluded because with few exceptions it did not generate any significant effects in the first analysis.[1] This decision was made in order to facilitate analysis of results. Instead of sex being used in analysis two as a grouping factor, Exp/Control was substituted which differentiated the experimental from the control group. The second analysis elaborates on the first in that a comparison of mean reaction times, physiological responses and moods was available for the two reaction time tasks (the flashing rectangle and the stop-sign). This generated a test of the assumption that reaction time at a

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1. There were two exceptions where sex exerted a significant effect on the results of analysis one. In the pre-caffeine systolic blood pressure phase and the pre-post diastolic blood pressure difference phase, a significant effect occurred with sex. Large standard deviations characterised both of these cases.

specific caffeine level would be mirrored when the rectangle was replaced by the stop-sign at the same dosage. The pre-post difference measures express the measures after caffeine is administered as a proportion of the pre-test measure. A table of the significant results for analysis two follows, accompanied by a discussion of these.

TABLE 2. BMDP2V UNEQUAL CELLS ANALYSIS TWO.

	F	TAIL PROBABILITY
1. PRE-DOSE SYSTOLIC BLOOD PRESSURE		
EXP/CONTROL	4.50	0.424
SESSION, EXP/CONTROL	7.77	0.0091
2. PRE-DOSE DIASTOLIC BLOOD PRESSURE		
SESSION	7.20	0.0117
3. PRE-DOSE MEAN REACTION TIME		
PERSONALITY	4.54	0.0418
SESSION	9.14	0.0052
PERSONALITY, SESSION, EXP/CONTROL	6.55	0.0159
4. PRE-DOES MOOD 2 (NORMAL BREATHING/BREATHLESS)		
SESSION, PERSONALITY	4.24	0.0482
5. PRE-DOSE MOOD 3 (SLEEPY/ALERT)		
SESSION,EXPT/CONT	4.68	0.0385
6. PRE-POST SYSTOLIC BLOOD PRESSURE DIFFERENCE		
PERSONALITY,EXPT/CONT	4.63	0.0395
7. PRE-POST MEAN REACTION TIME DIFFERENCE		
EXP/CONTROL	4.90	0.0349

1. ANALYSIS TWO

1. Figure (21) demonstrates a significant effect with the Exp/Control variable for the pre-dose phase of systolic blood pressure. The experimental group had lower pre-dose systolic blood pressures than the control group. There was also a significant sessions x exp/control interaction effect which is also demonstrated in figure (21). The control group showed a decrement in pre-dose systolic blood pressure on the second session (the stop sign) compared with session one (the flashing rectangle). The experimental group exhibited a trend in the opposite direction in which pre-dose systolic blood pressure increased from the first to the second session.

2. There was also a significant sessions effect for pre-dose diastolic blood pressure which is demonstrated in figure (22). A uniform decrease in diastolic blood pressure occurred on the second session for each of the experimental groups. The trend was strongest for the control group, however, this did not reach a level of significance.

3. Pulse demonstrated no significant pre-dose effects, however, mean reaction time demonstrated several. See figure (24). Although personality type and sessions effects occurred with pre-dose phase mean reaction time, they were more appropriately

considered in the light of the significant personality type x sessions x exp/control interaction. Figure (24) shows that both introverted and extroverted control group members tended to have shorter mean reaction times for the second session than the first. This trend was also followed by the introvert experimental group but not by the extroverted experimental group members, who demonstrated a trend in the opposite direction. The largest change in mean reaction time between sessions occurred for the introverted experimental group, followed by extrovert controls, introvert controls and the extrovert experimental group.

4. Figure (26) demonstrates a significant sessions x personality type interaction effect on the pre-dose phase of mood 2 (Normal breathing/Breathless). Introverts demonst ıte a higher level of breathlessness on the initial session (flashing rectangle) than on the second session (the stop sign), while the trend for extroverts is in the opposite direction with higher levels of breathlessness on the second session than the first, although the difference is small.

5. Figure (27) demonstrates a significant sessions x exp/control interaction effect for the pre-dose phase of mood 3 (Sleepy/Alert). The members of the experimental group tended to be more alert on the initial session than they were on the second .

session. The opposite occurred for the control group in which higher levels of alertness were obtained on the second session.

2. PRE-POST DOSE DIFFERENCES FOR ANALYSIS TWO

1. There were only two significant pre-post difference effects in analysis two, the first of which was pre-post systolic blood pressure difference. These differences were obtained using the same method used for obtaining the pre-post caffeine differences in analysis one. Figure (31) depicts a significant personality type x exp/control interaction effect on the pre-post systolic blood pressure difference phase. The initial session pre-post systolic blood pressure difference was positive for all groups, except the extrovert experimental group and the movement for all three groups (excluding extrovert experimentals) followed a negative trend to the second session difference. The extrovert experimental group, as demonstrated in figure (31) had a slightly negative initial pre-post difference which became a positive pre-post systolic blood pressure difference in the second session.

2. Figure (33) depicts a significant exp/control effect on the pre-post mean reaction time difference phase. There was a uniform trend demonstrated by both the control group and the experimental

group in which the pre-post mean reaction time difference for the initial phase is more positive than for the second phase. This trend is significantly more pronounced for the experimental group than it is for the control group.

There are no further pre-post differences demonstrated on any of the figures for any of the six mood characteristics.

Figure (1)

The Means and Standard Deviations of Systolic Blood Pressure at the three pre-treatment levels for each separate category.

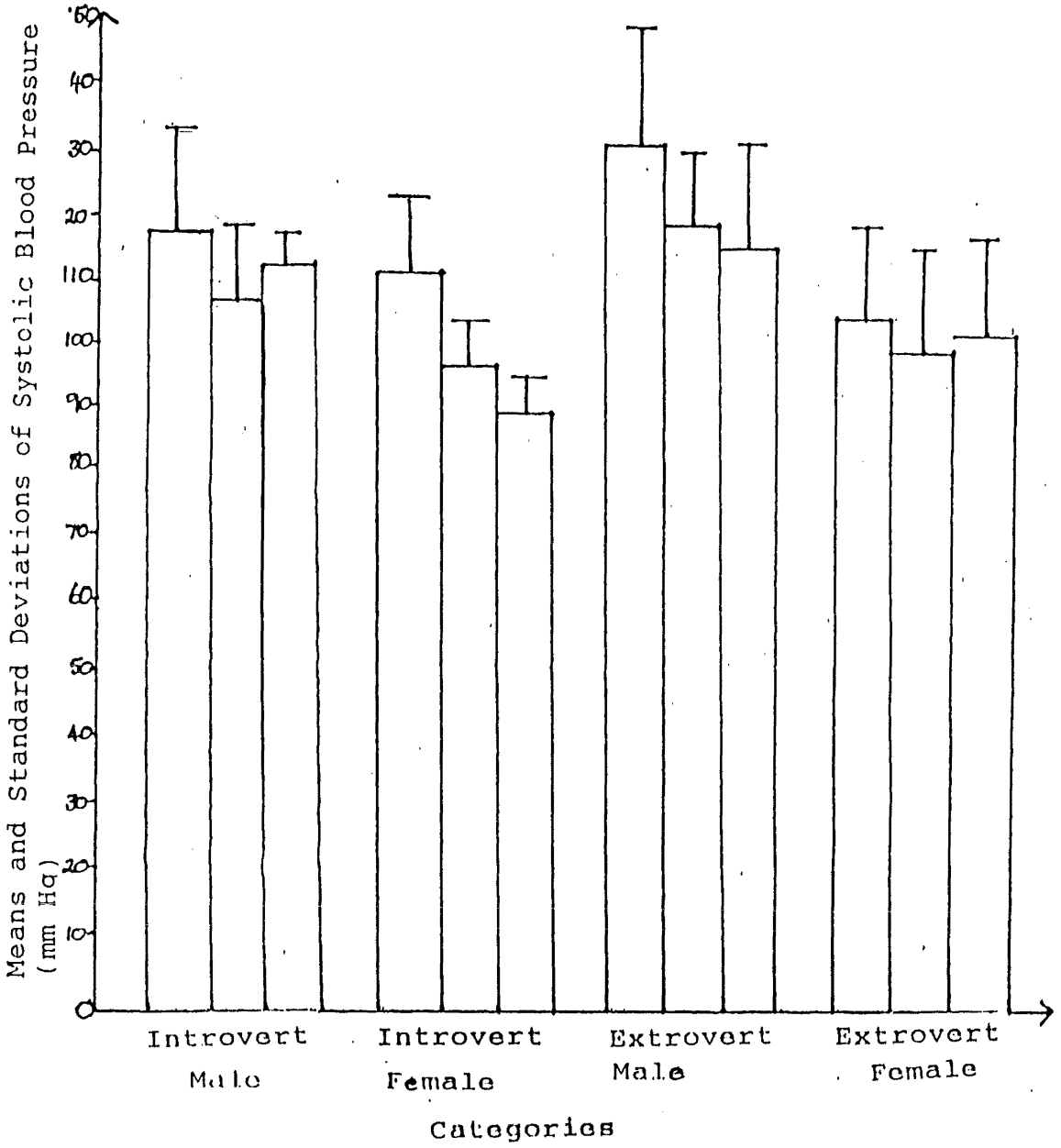


Figure (2)

The Means and Standard Deviations of Diastolic Blood Pressure at the three pre-treatment levels for each separate category.

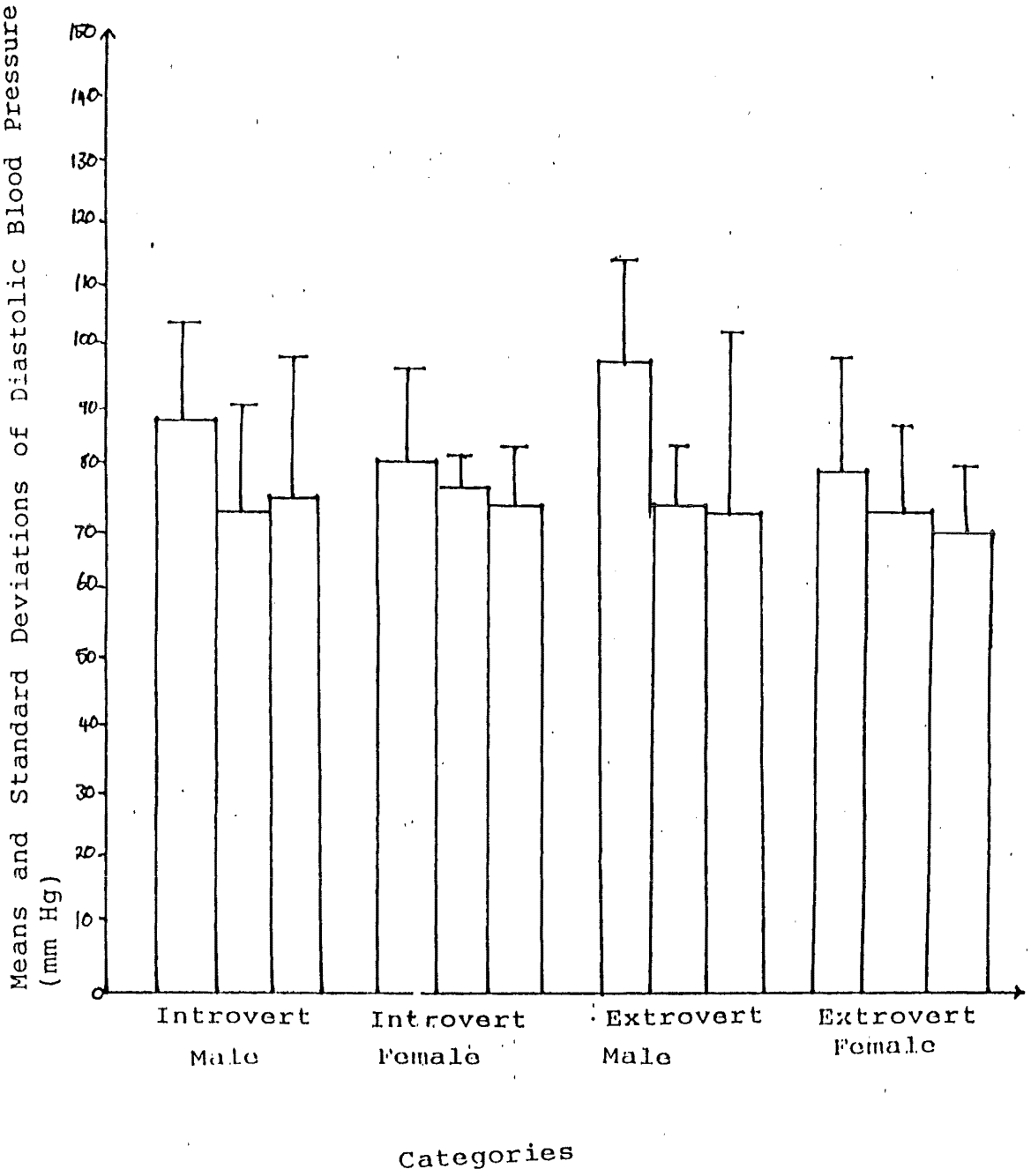


Figure (3)

The Means and Standard Deviations of Pulse Rate at the three pre-treatment levels for each separate category.

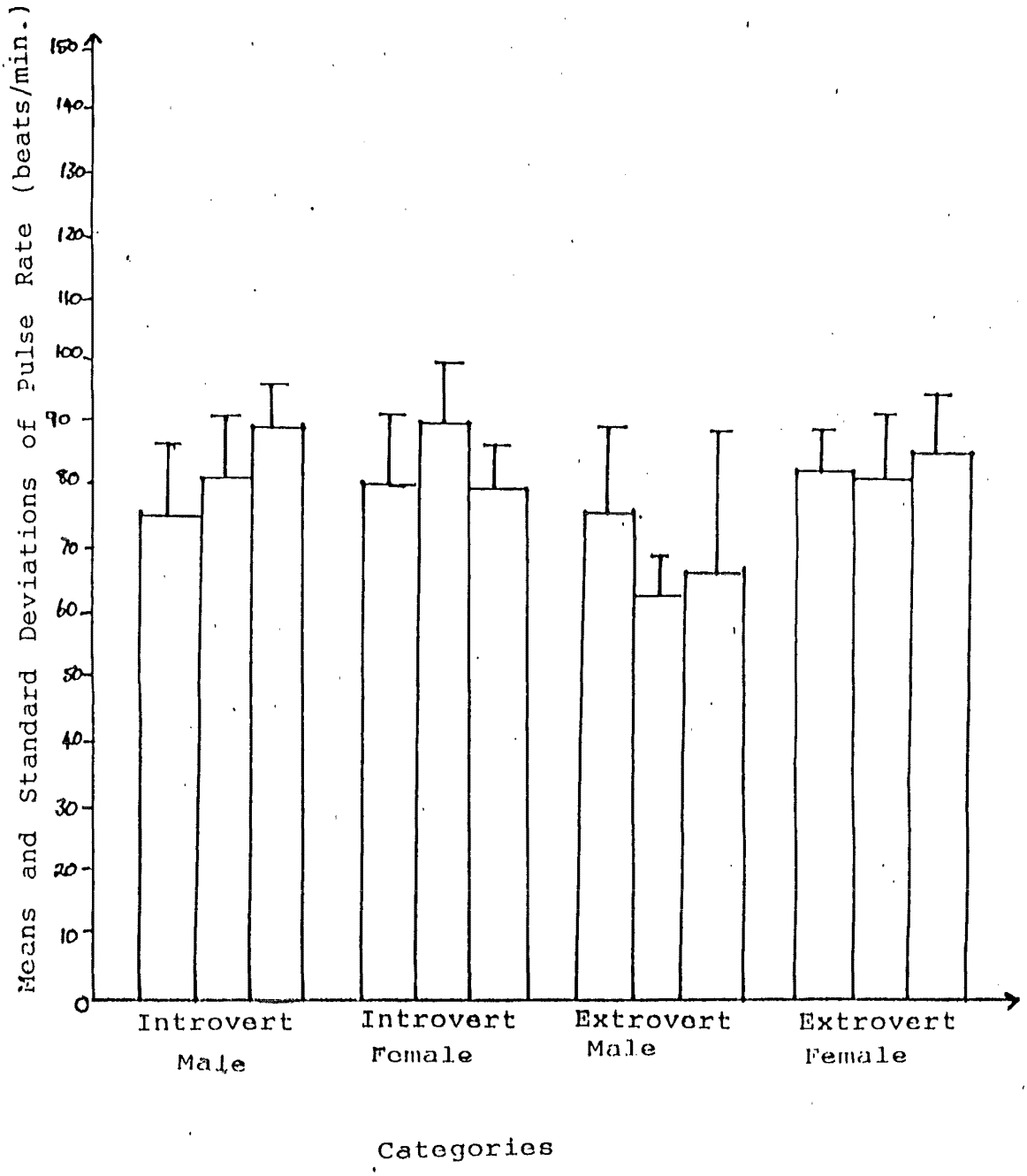


Figure (4)

The Means and Standard Deviations of Mean Reaction Times at the three pre-treatment levels for each separate category.

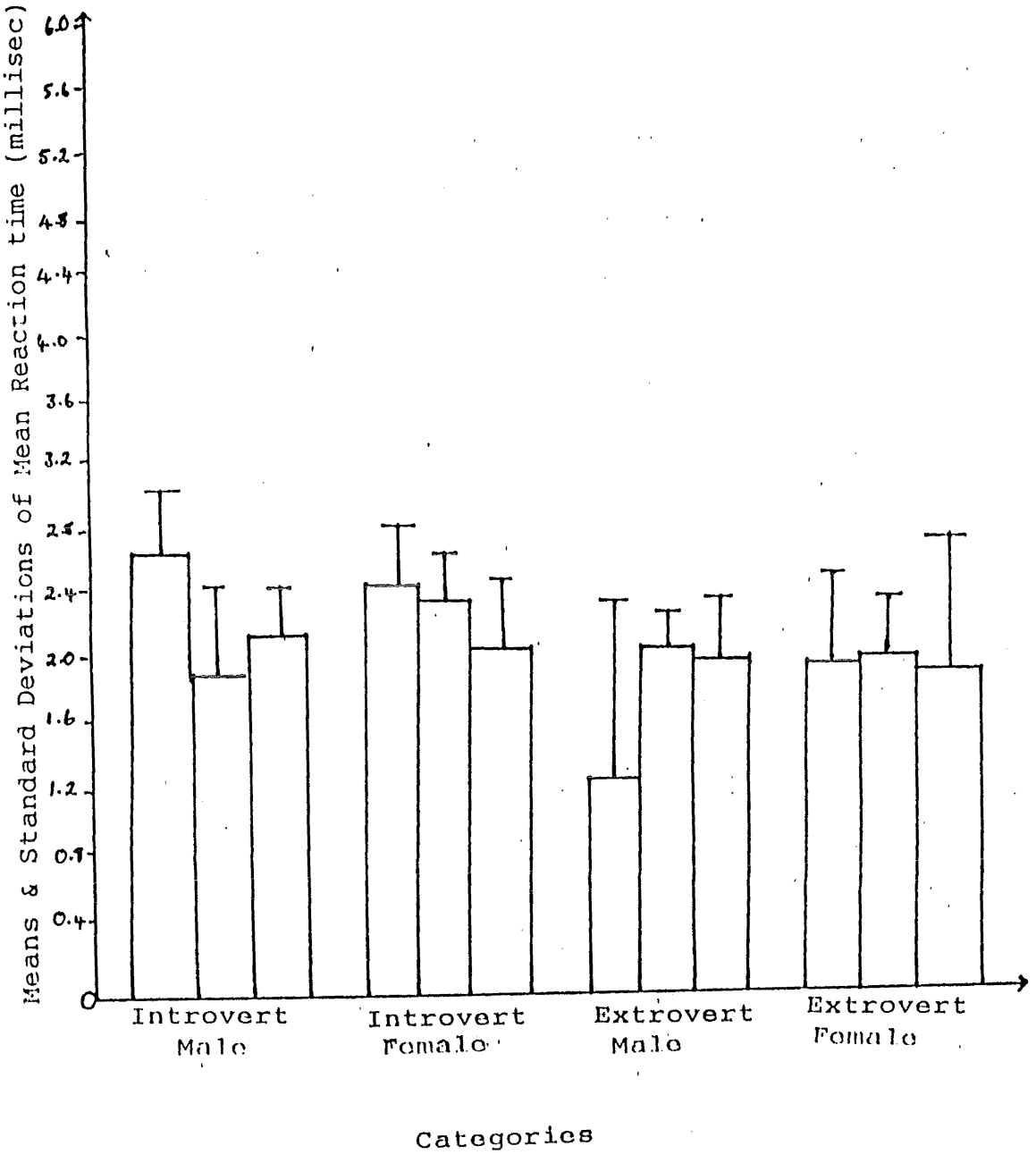


Figure (5)

The Means and Standard Deviations of Mood 1 (Relaxed/Restless) at the three pre-treatment levels for each separate category.

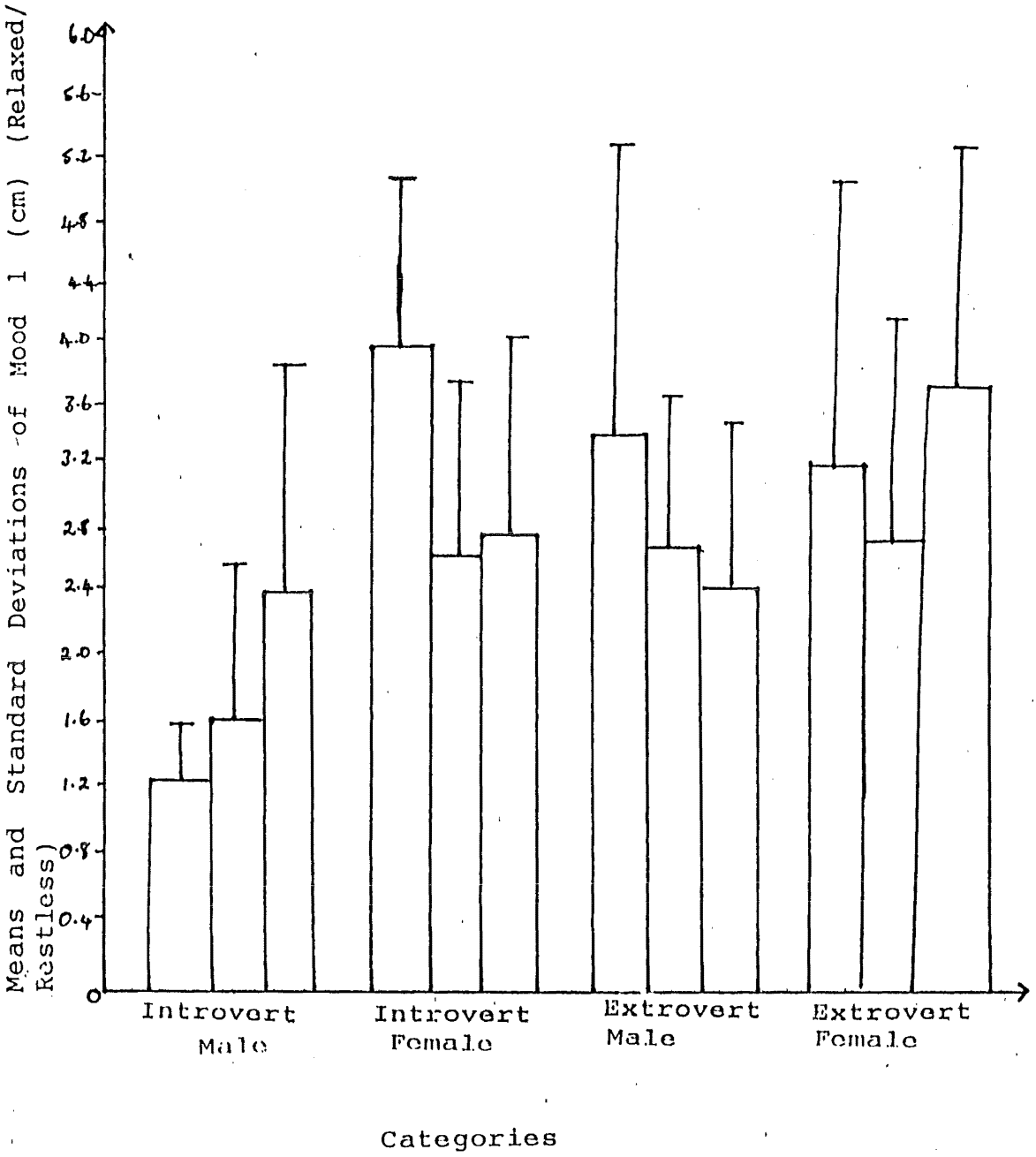


Figure (6).
The means and standard deviations of mood 2 (normal breathing/breat-lessness) at the three pre-treatment levels for each separate category.

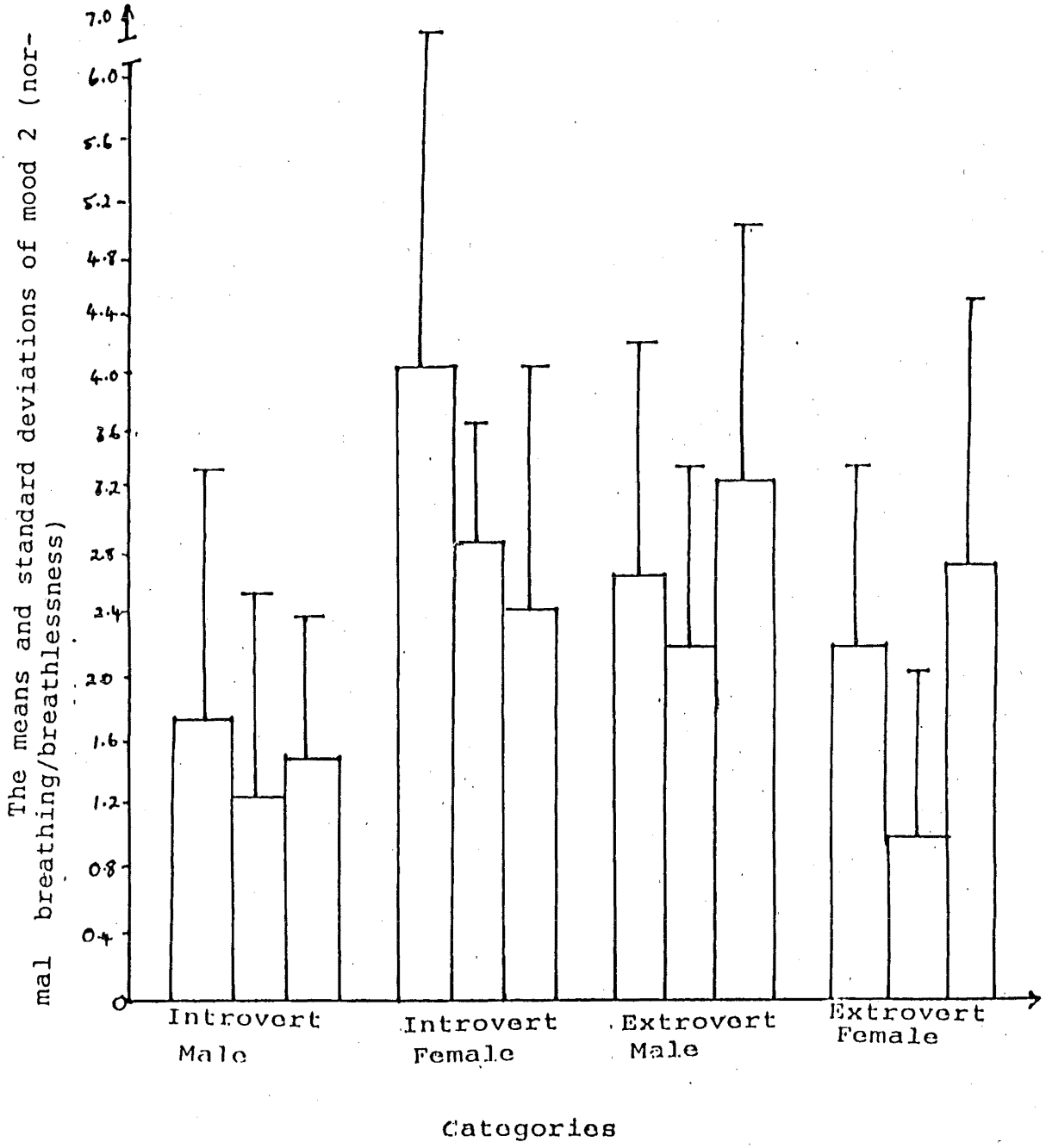


Figure (7)

The Means and Standard Deviations of Mood 3 (Sleepy/Alert) at the three pre-treatment levels for each separate category.

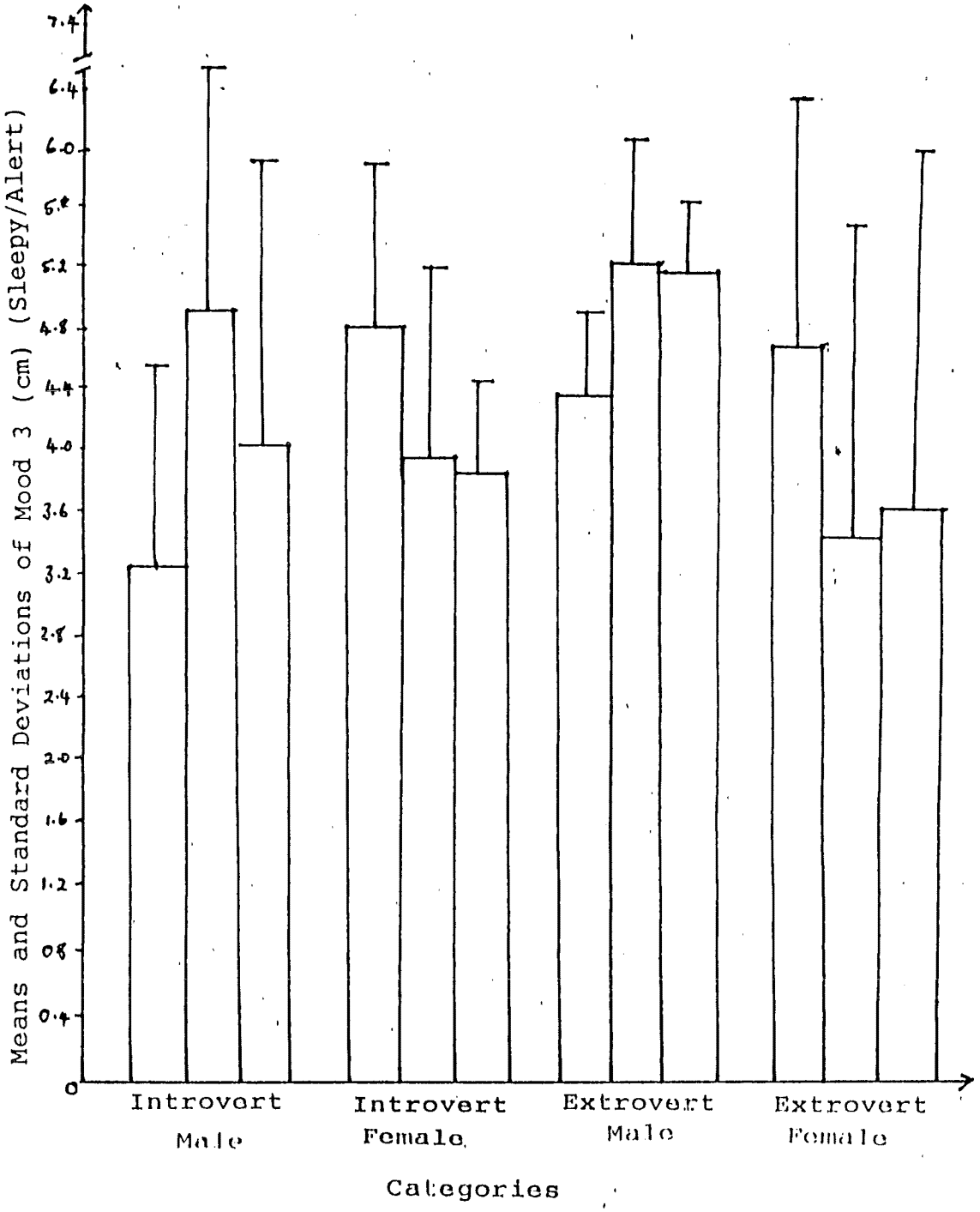


Figure (8)

The Means and Standard Deviations of Mood 4 (Unhappy/happy) at the three pre-treatment levels for each separate ategory.

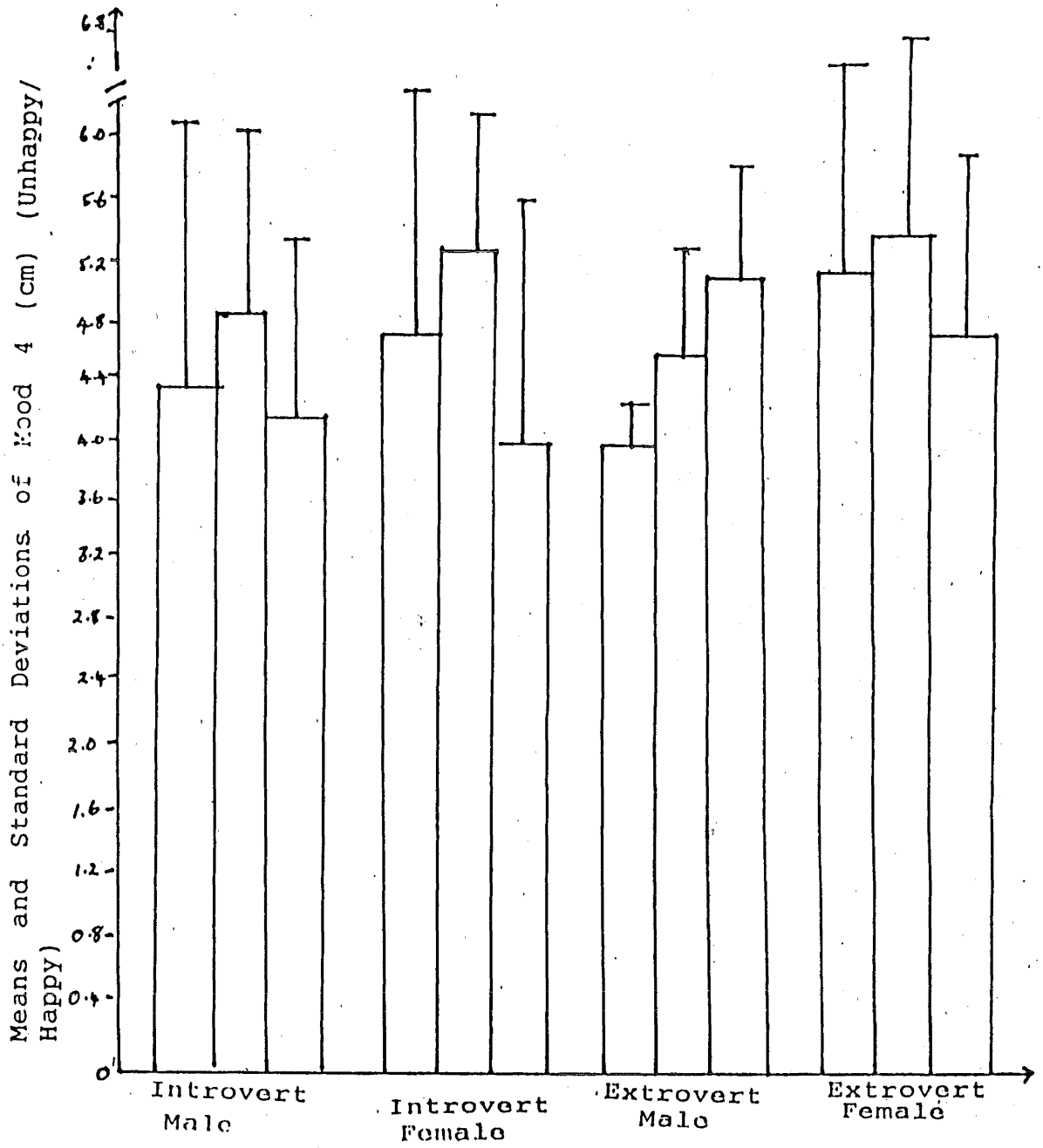


Figure (9)

The Means and Standard Deviations of Mood 5 (Calm/Nervous) at the three pre-treatment levels for each separate category.

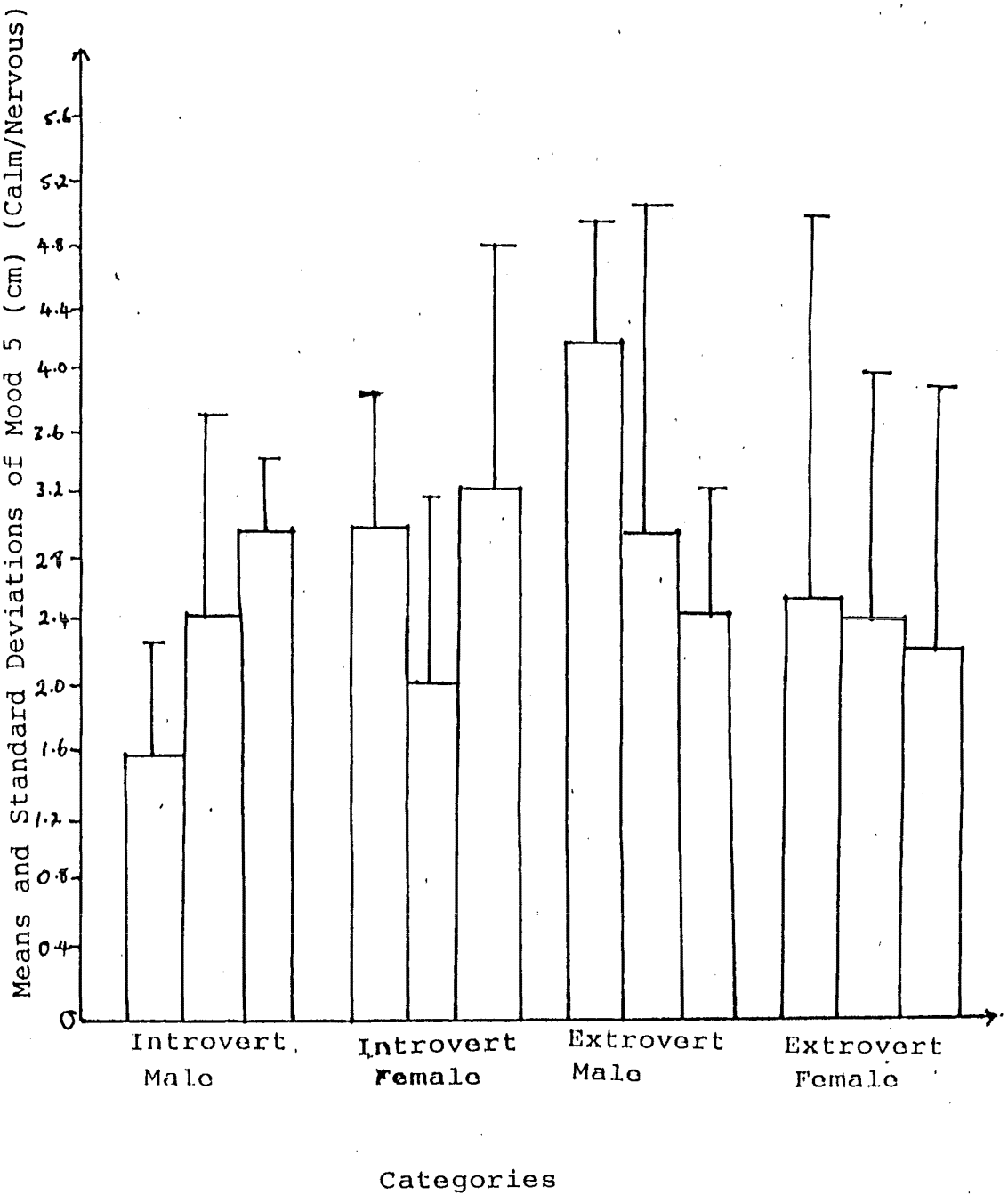


Figure (10)

The Means and Standard Deviations of Mood 6 (Confidence lacking/Confident) at the three pre-treatment levels for each separate category.

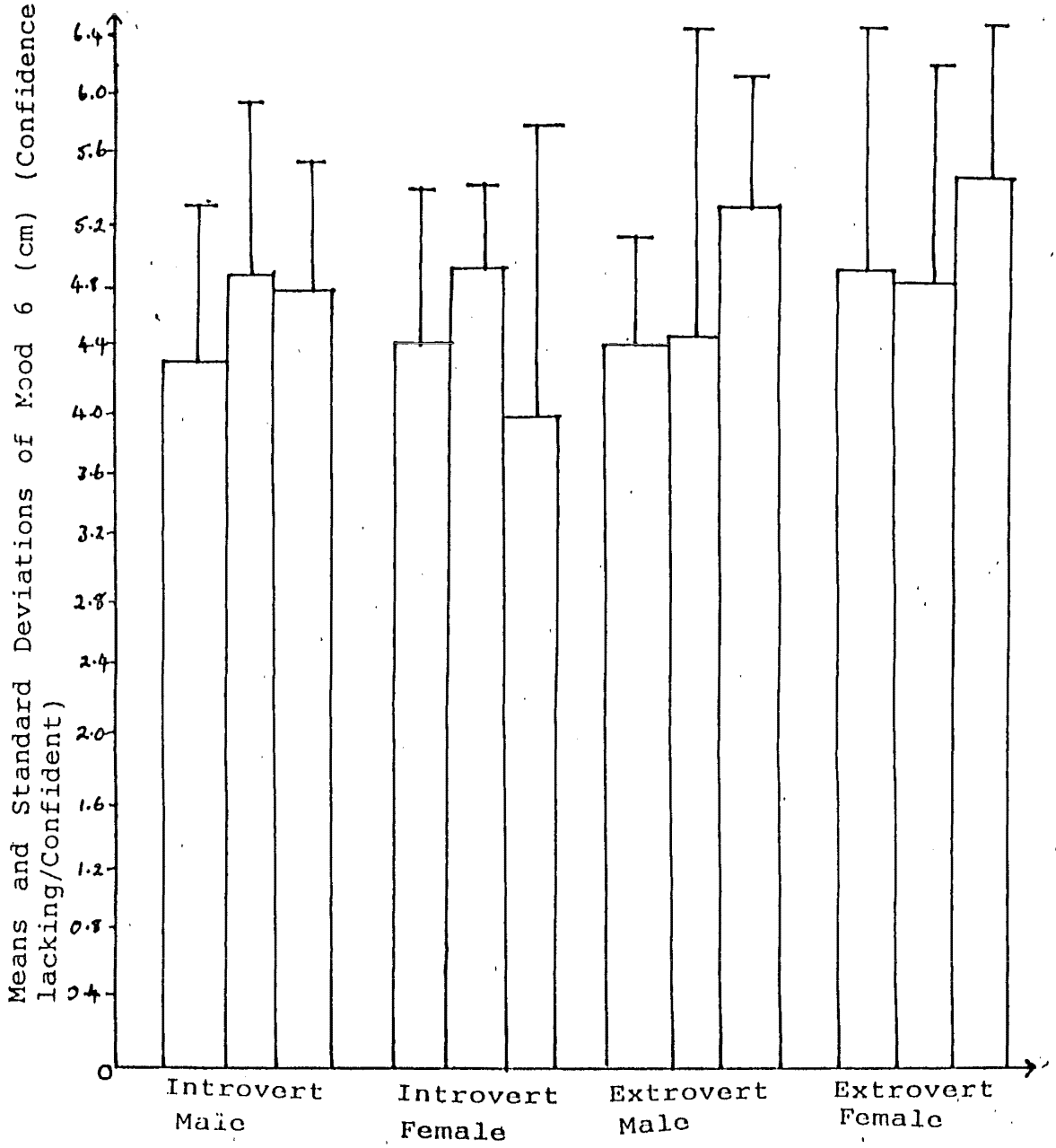


Figure (11)

The Means and Standard Deviations of Systolic Blood Pressure at the three Pre-Post treatment levels for each separate category.

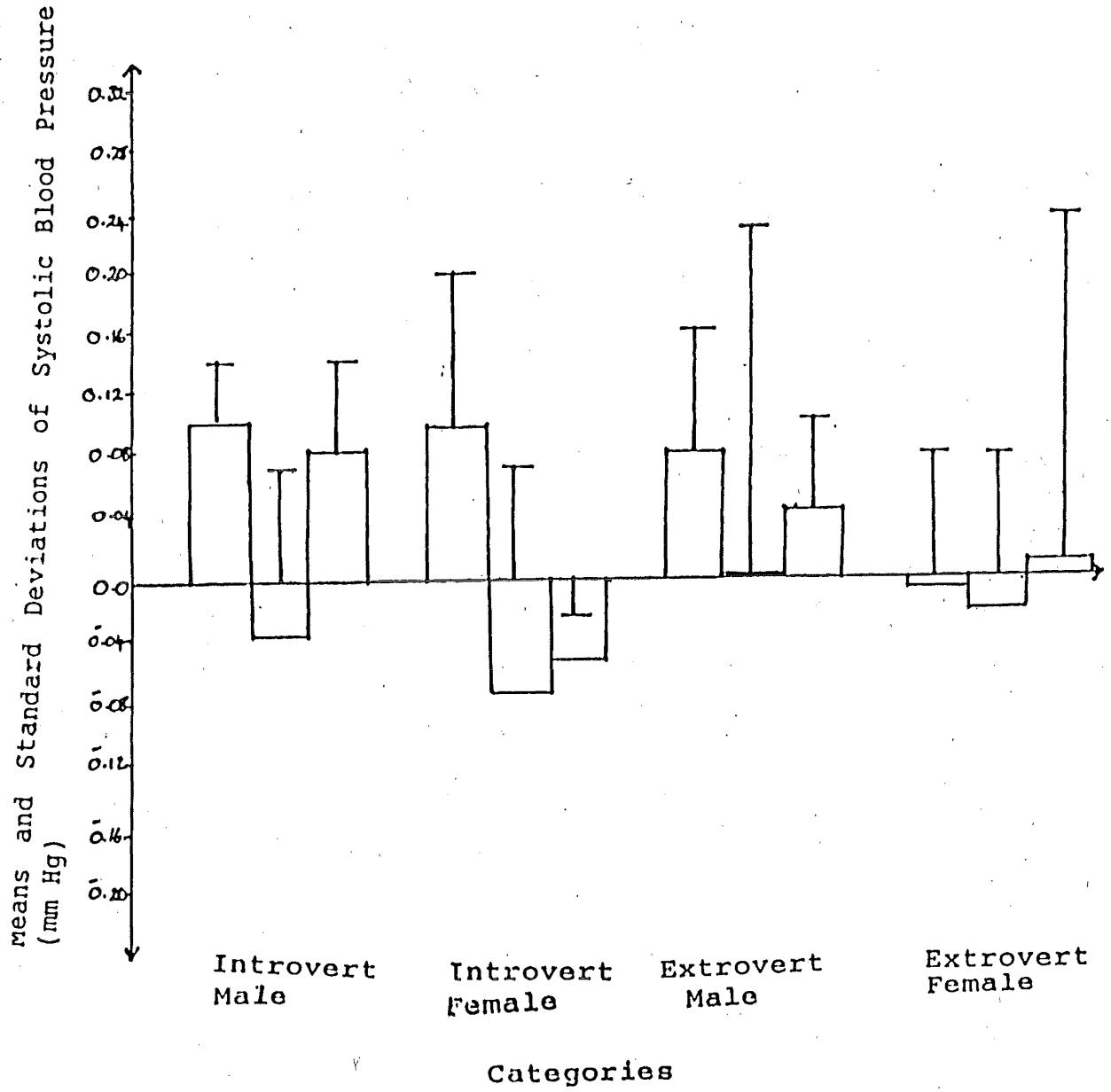


Figure (12)
The Means and Standard Deviations of Diastolic Blood Pressure at the three Pre-Post treatment levels for each separate category.

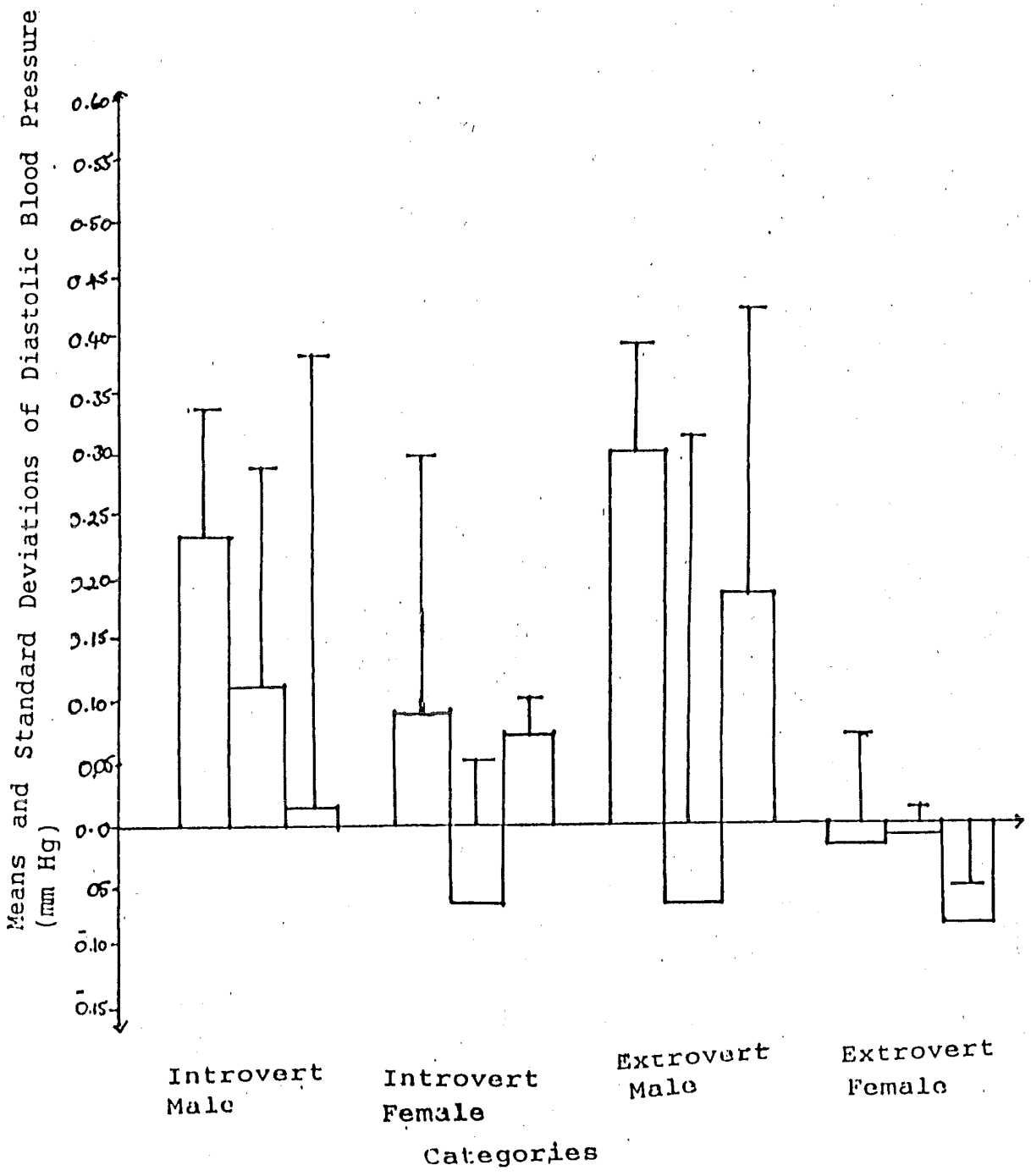


Figure (13)

The Means and Standard Deviations of Pulse Rate at the three Pre-Post treatment levels for each separate category.

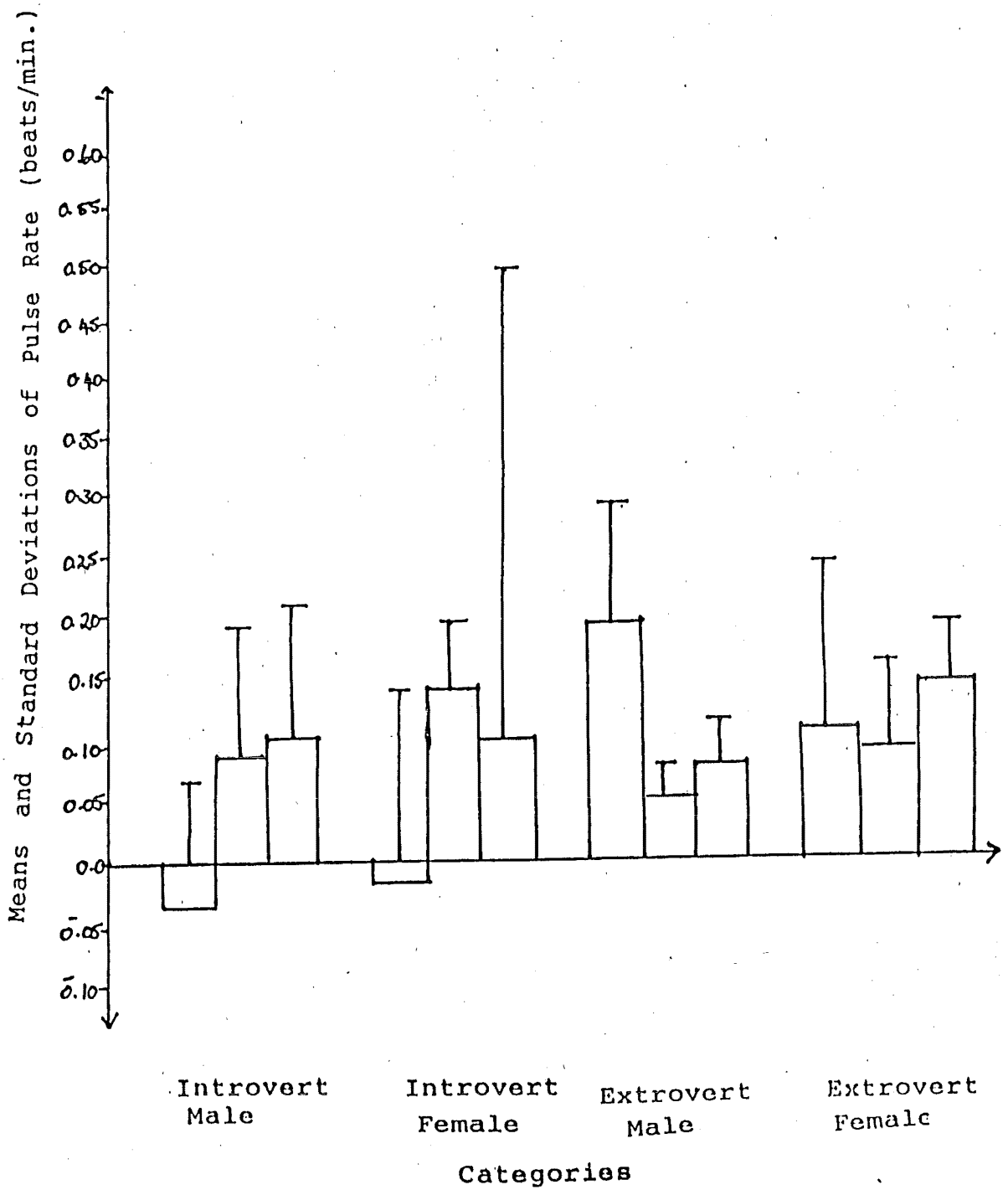


Figure (14)

The Means and Standard Deviations of Mean Reaction times at the three Pre-Post treatment levels for each separate category.

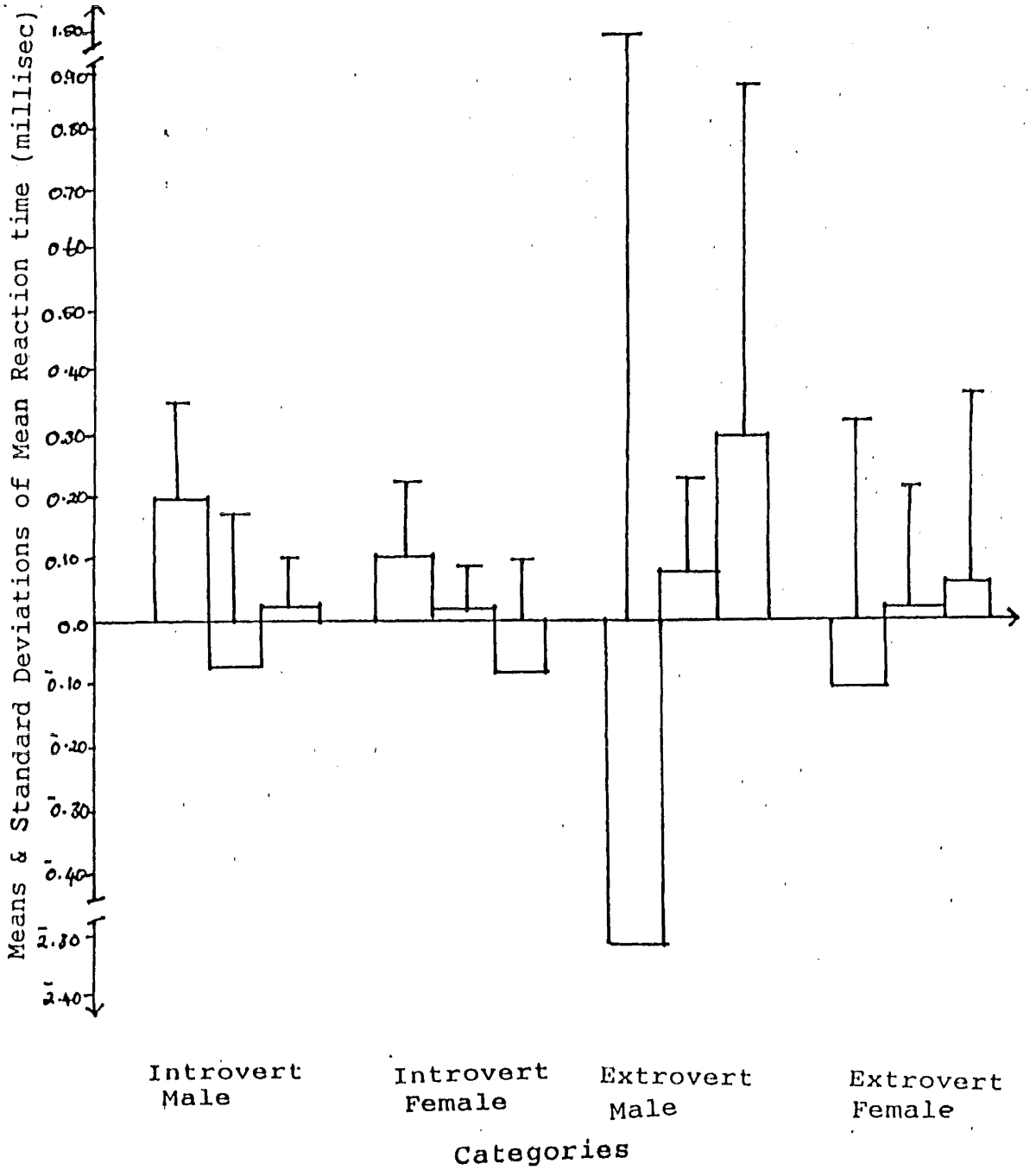


Figure (15)

The Means and Standard Deviations of Mood 1 (Relaxed/Restless) at the three Pre-Post treatment levels for each separate category.

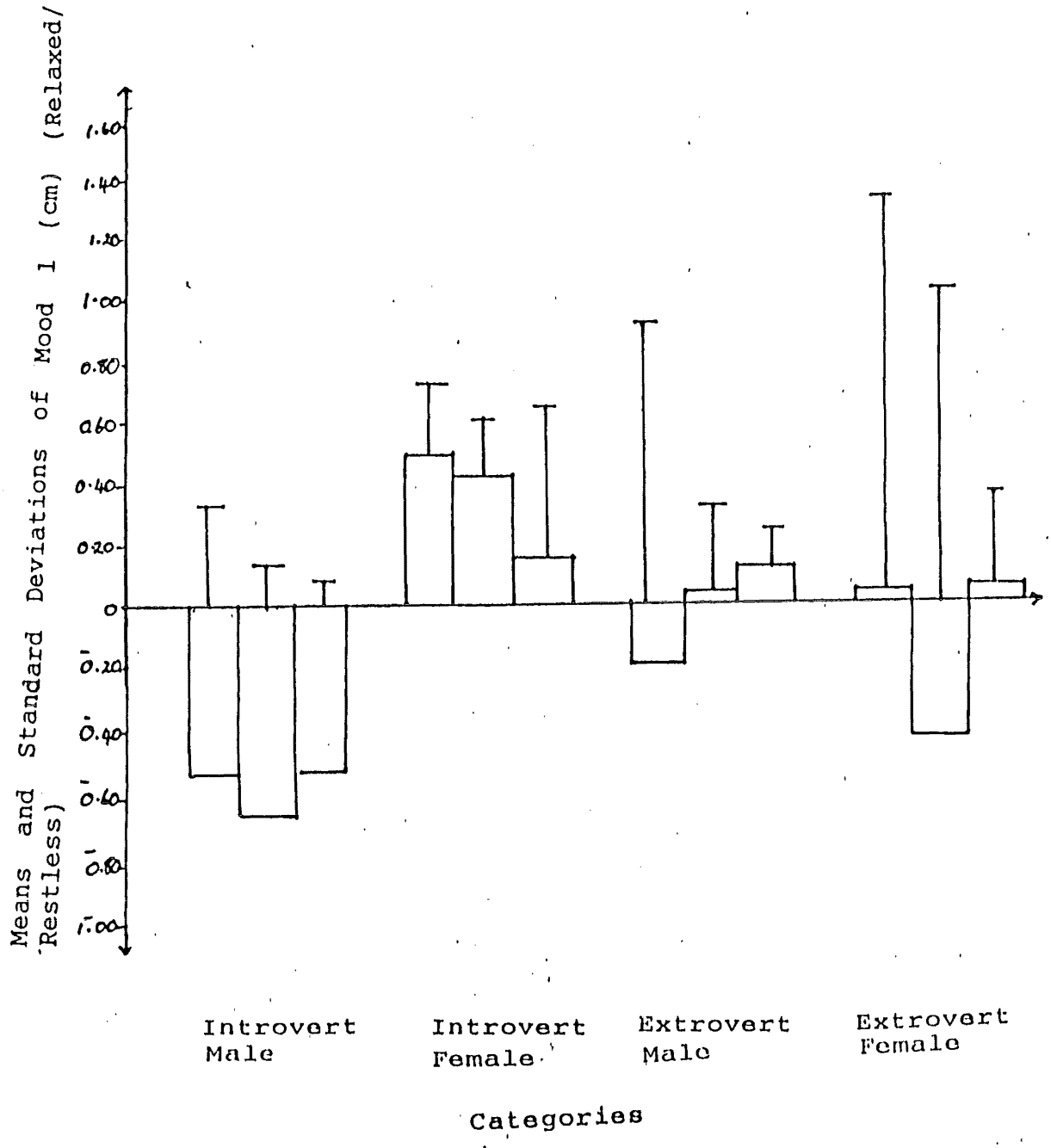


Figure (16) The means and standard deviations (normal breathing/breathlessness) at the 3 pre-post treatment levels for each separate category.

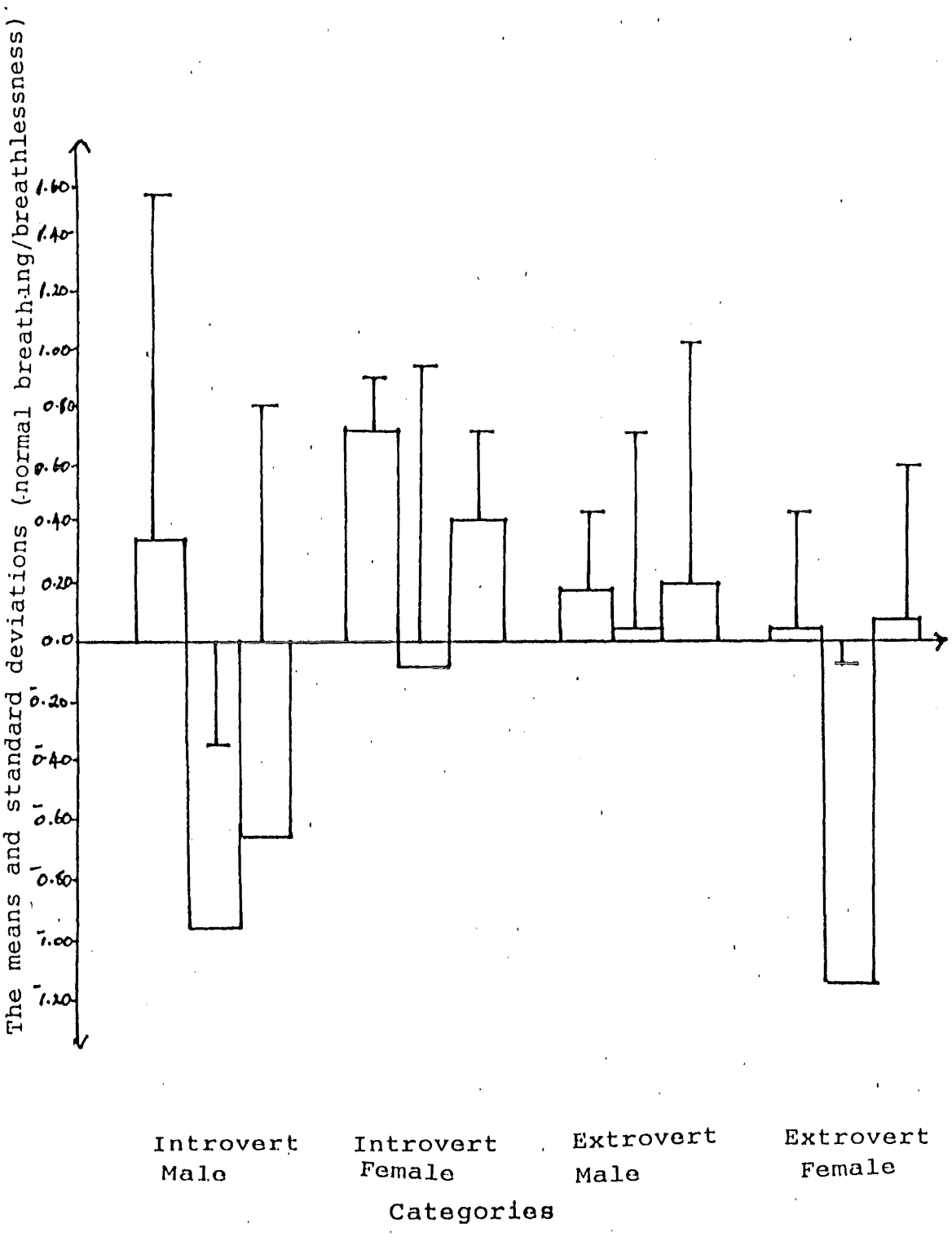


Figure (17)

The Means and Standard Deviations of Mood 3 (Sleepy/Alert) at the three Pre-Post treatment levels for each separate category.

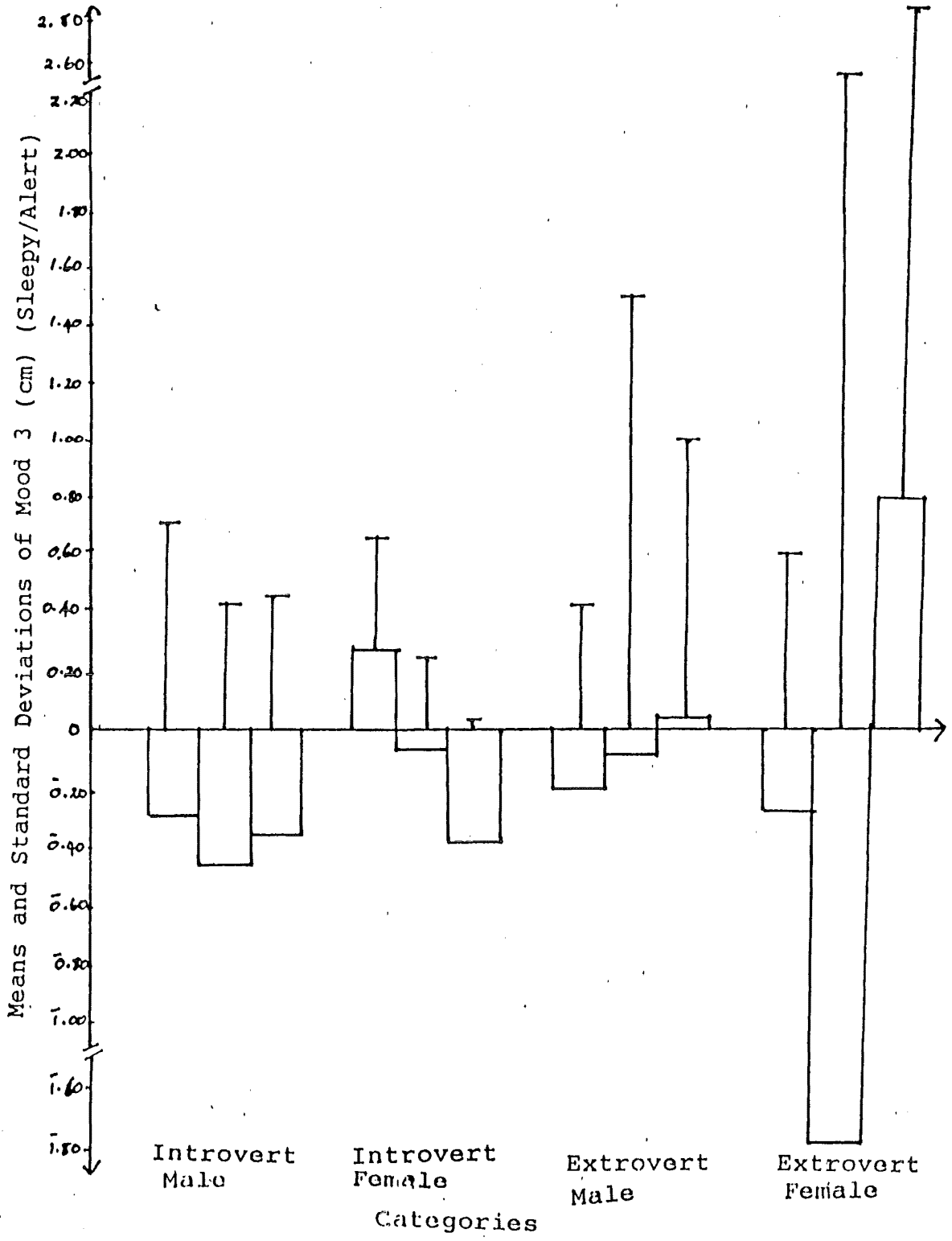


Figure (18)

The Means and Standard Deviations of Mood 4 (Unhappy/happy) at the three Pre-Post treatment levels for each separate category.

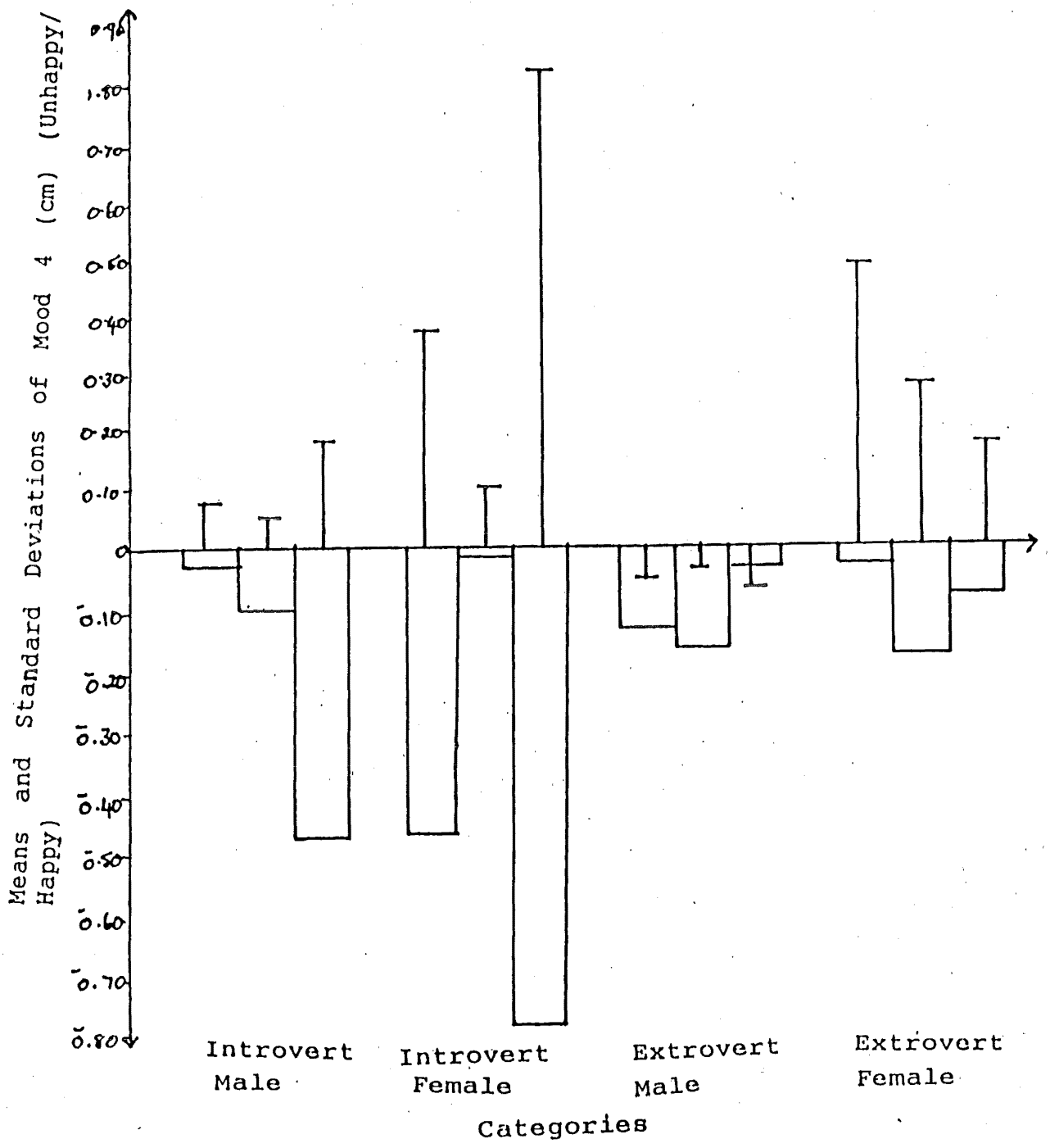


Figure (19)

The Means and Standard Deviations of Mood 5 (Calm/Nervous) at the three Pre-Post treatment levels for each separate category.

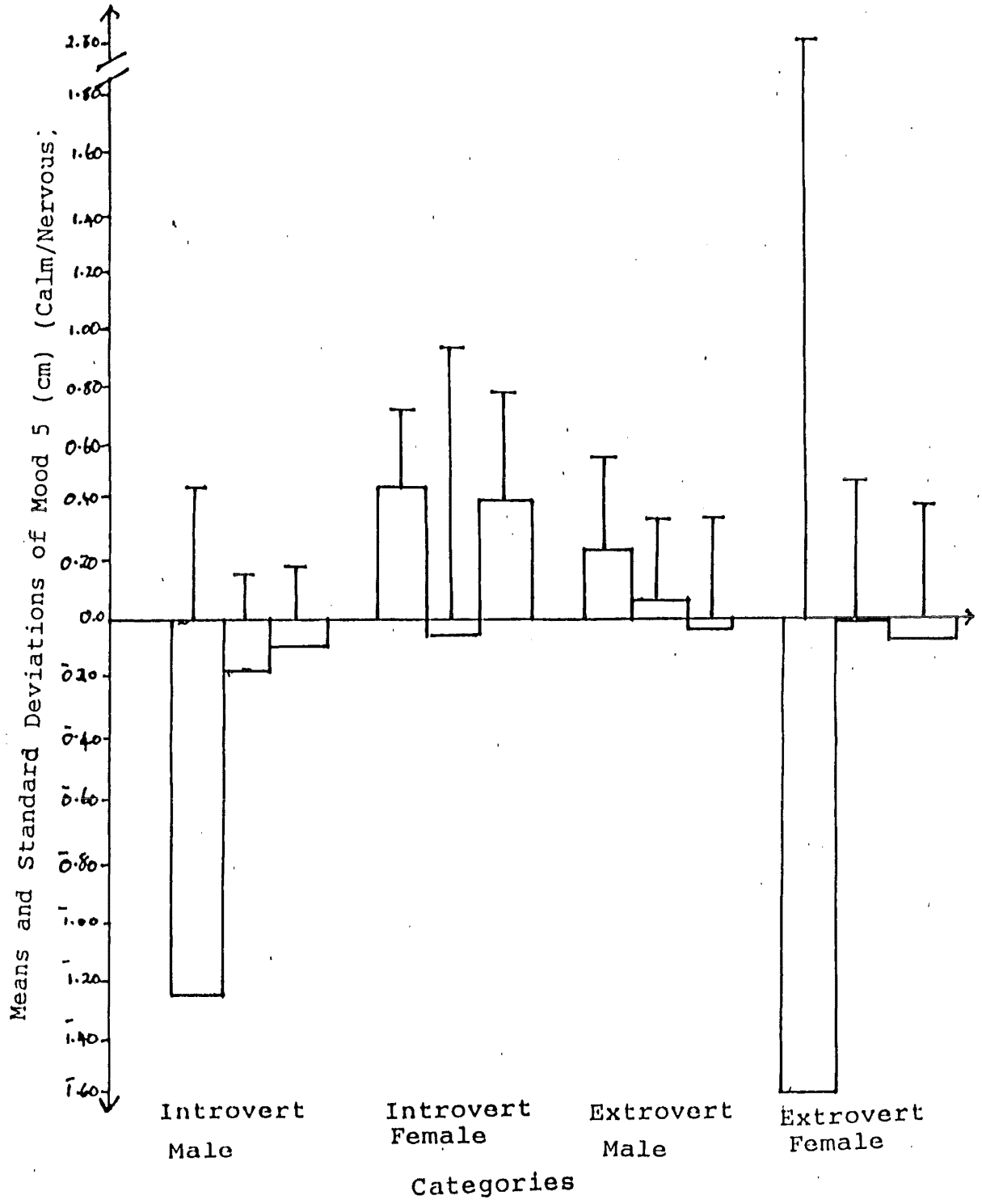


Figure (20)

The Means and Standard Deviations of Mood 6 (Confidence lacking/confident) at the three Pre-Post treatment levels for each separate category.

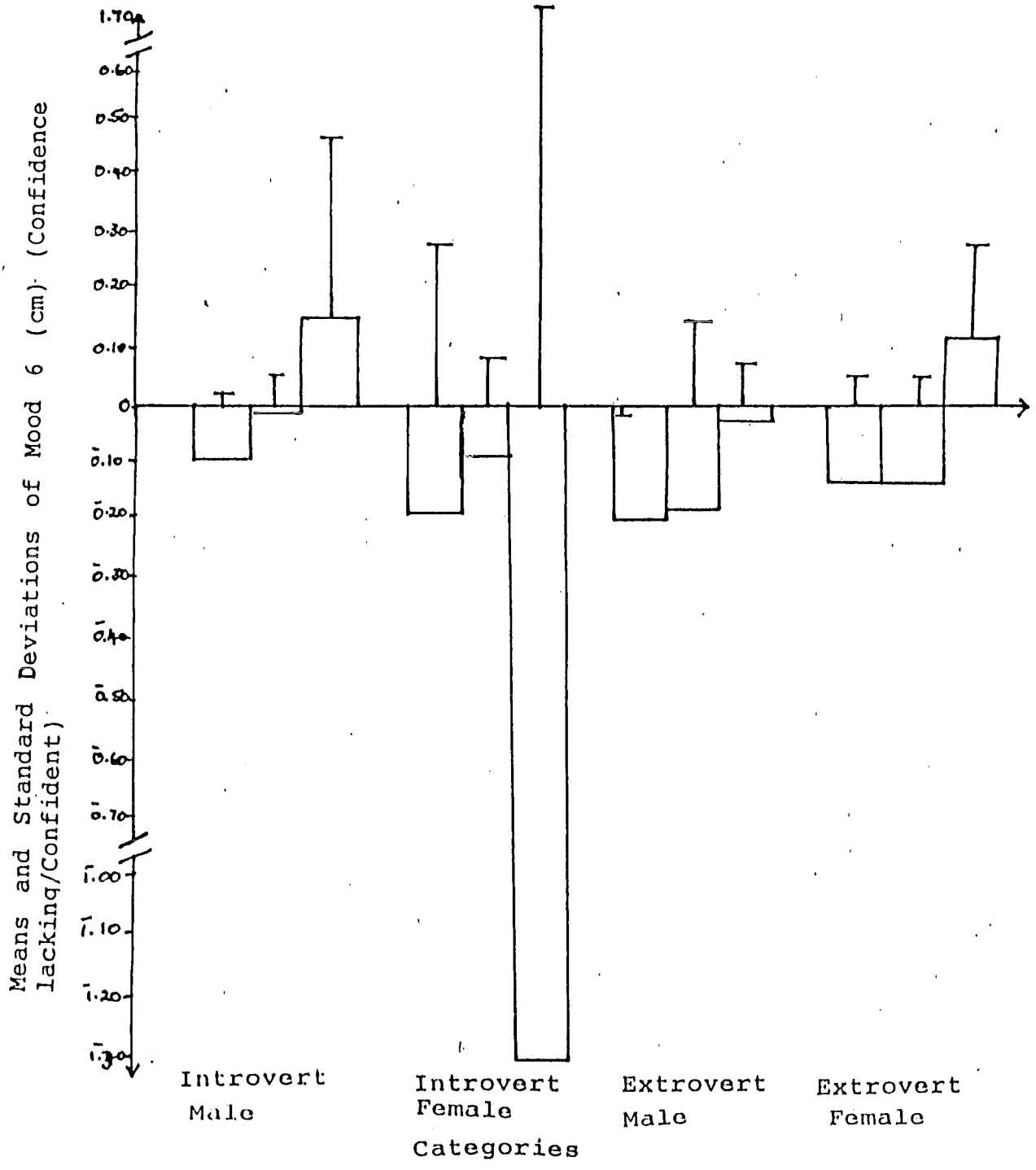


Figure (21) The Means and Standard Deviations of Systolic Blood Pressure at the two Pre-treatment levels for each separate category.

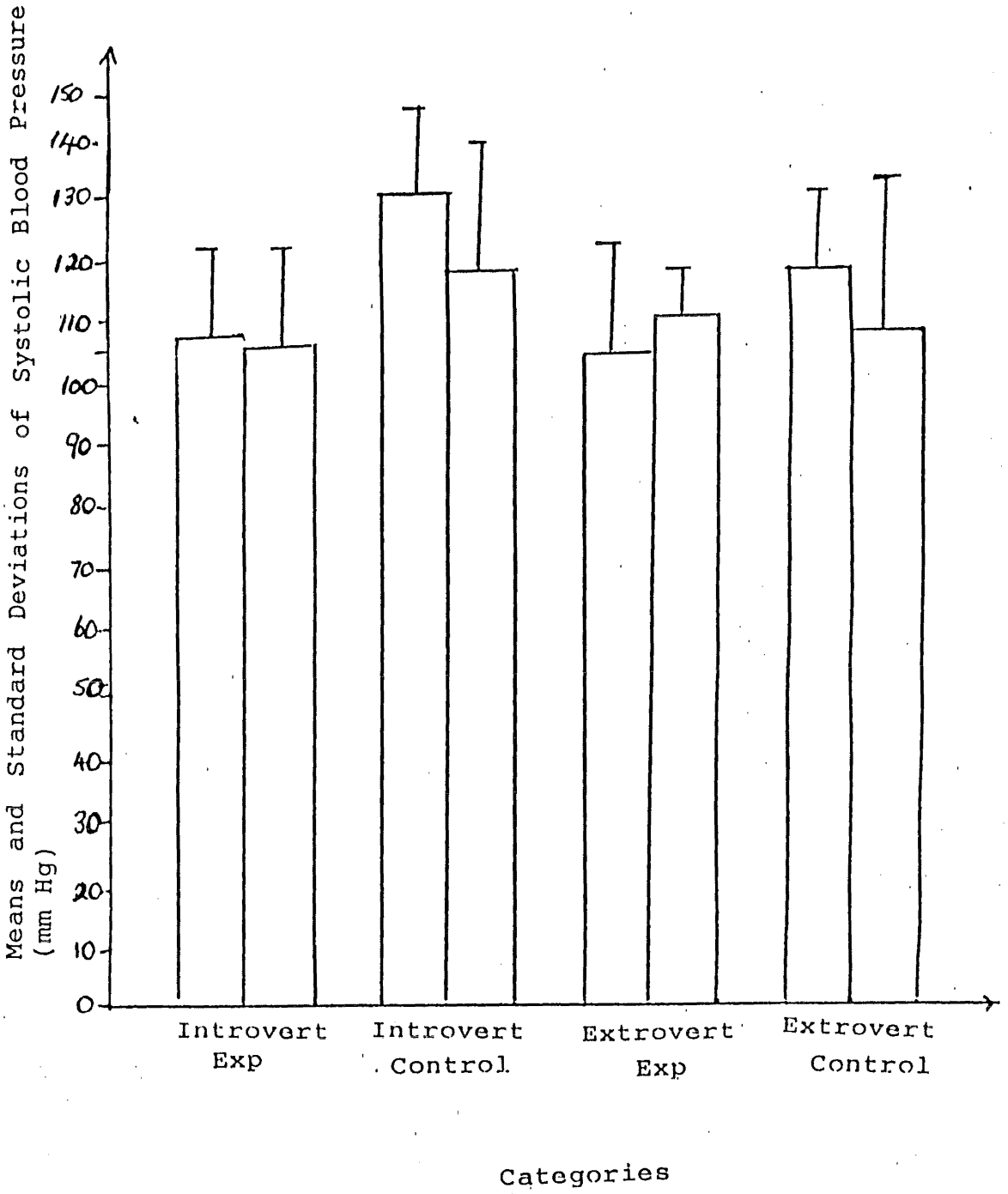


Figure (22) The Means and Standard Deviations of Diastolic Blood Pressure at the two Pre-treatment levels for each separate category.

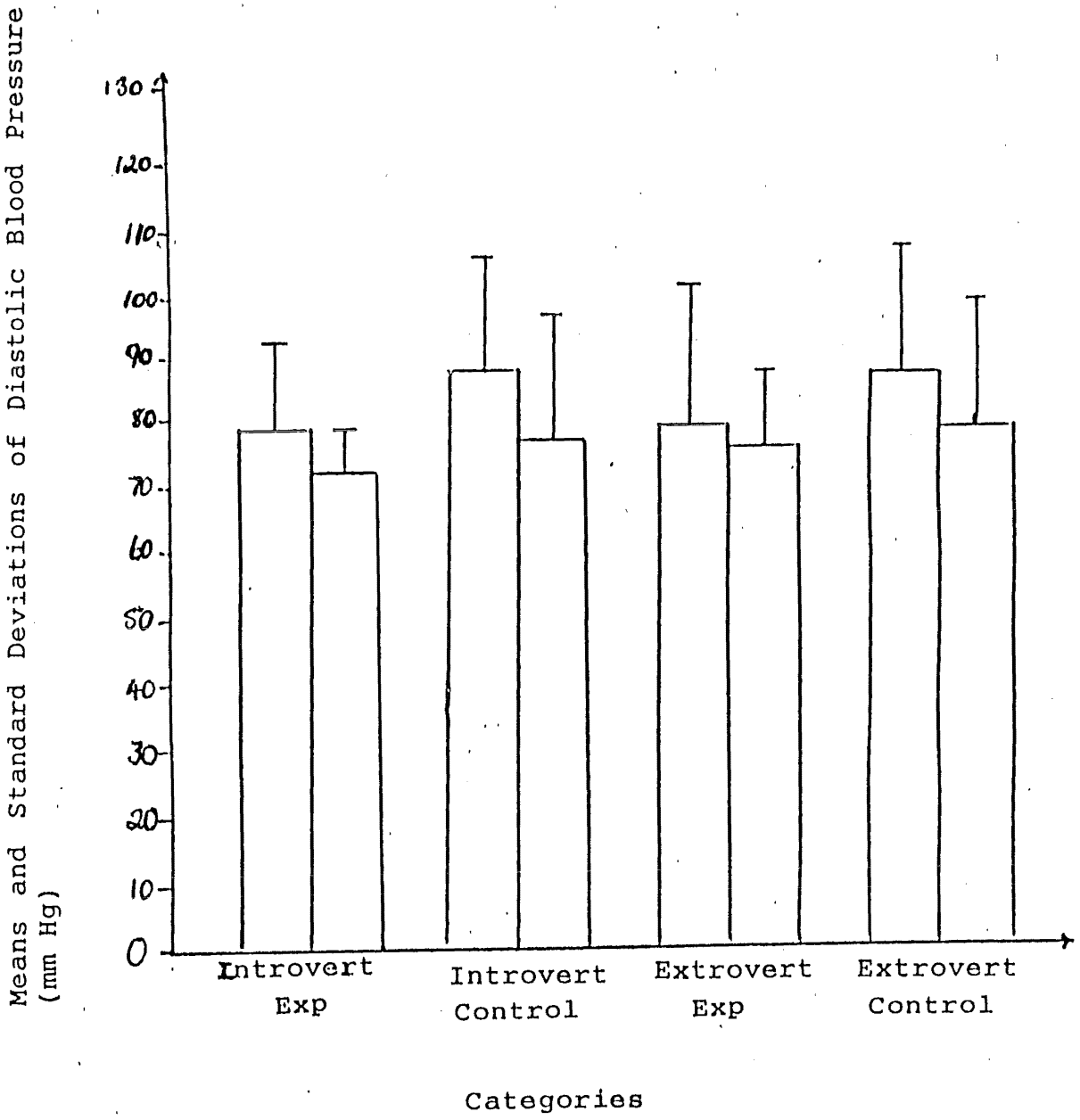


Figure (23)

The Means and Standard Deviations of Pulse Rate at the two Pre-treatment levels for each separate category.

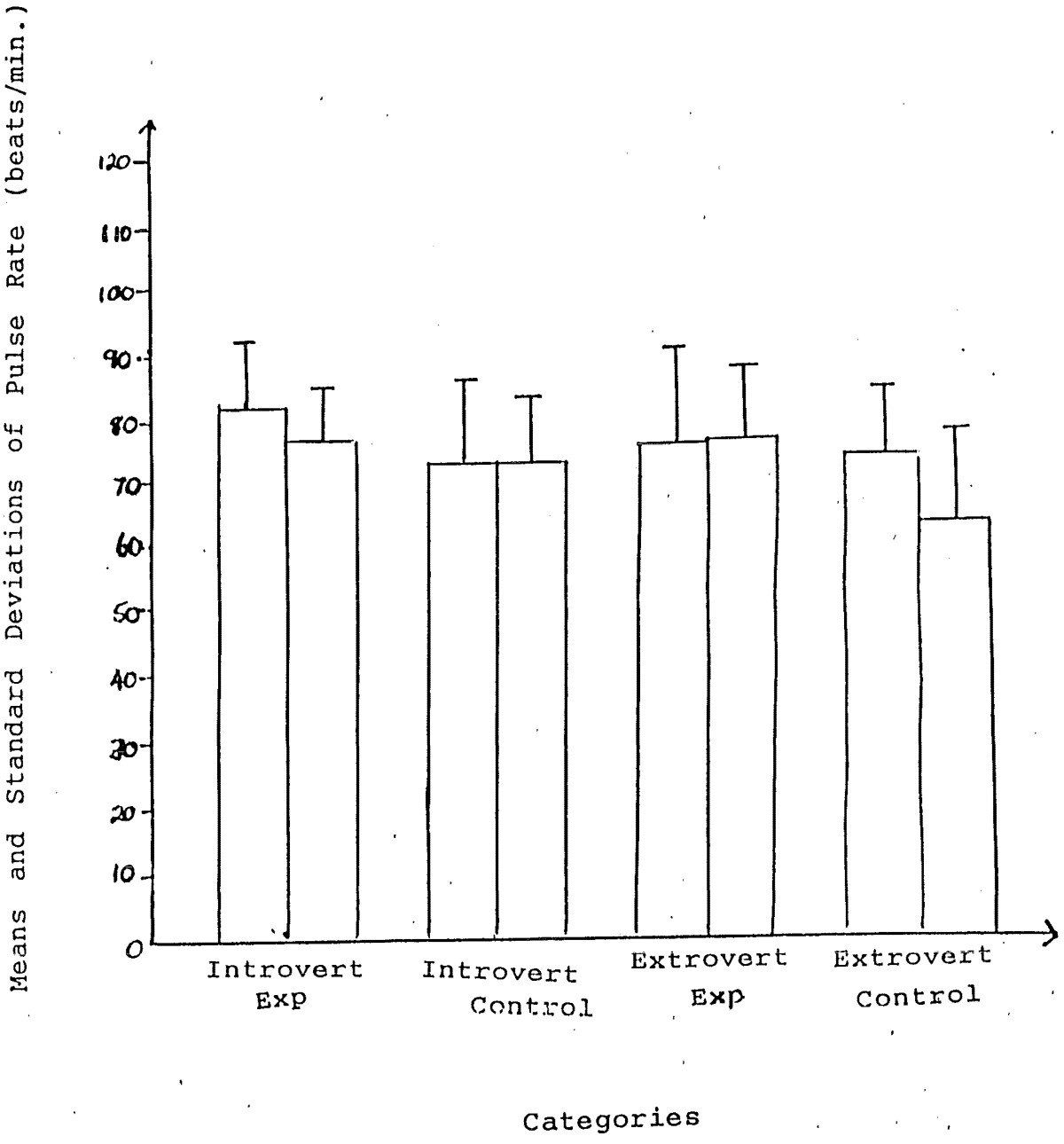


Figure (24)

The Means and Standard Deviations of Mean Reaction times at the two Pre-treatment levels for each separate category.

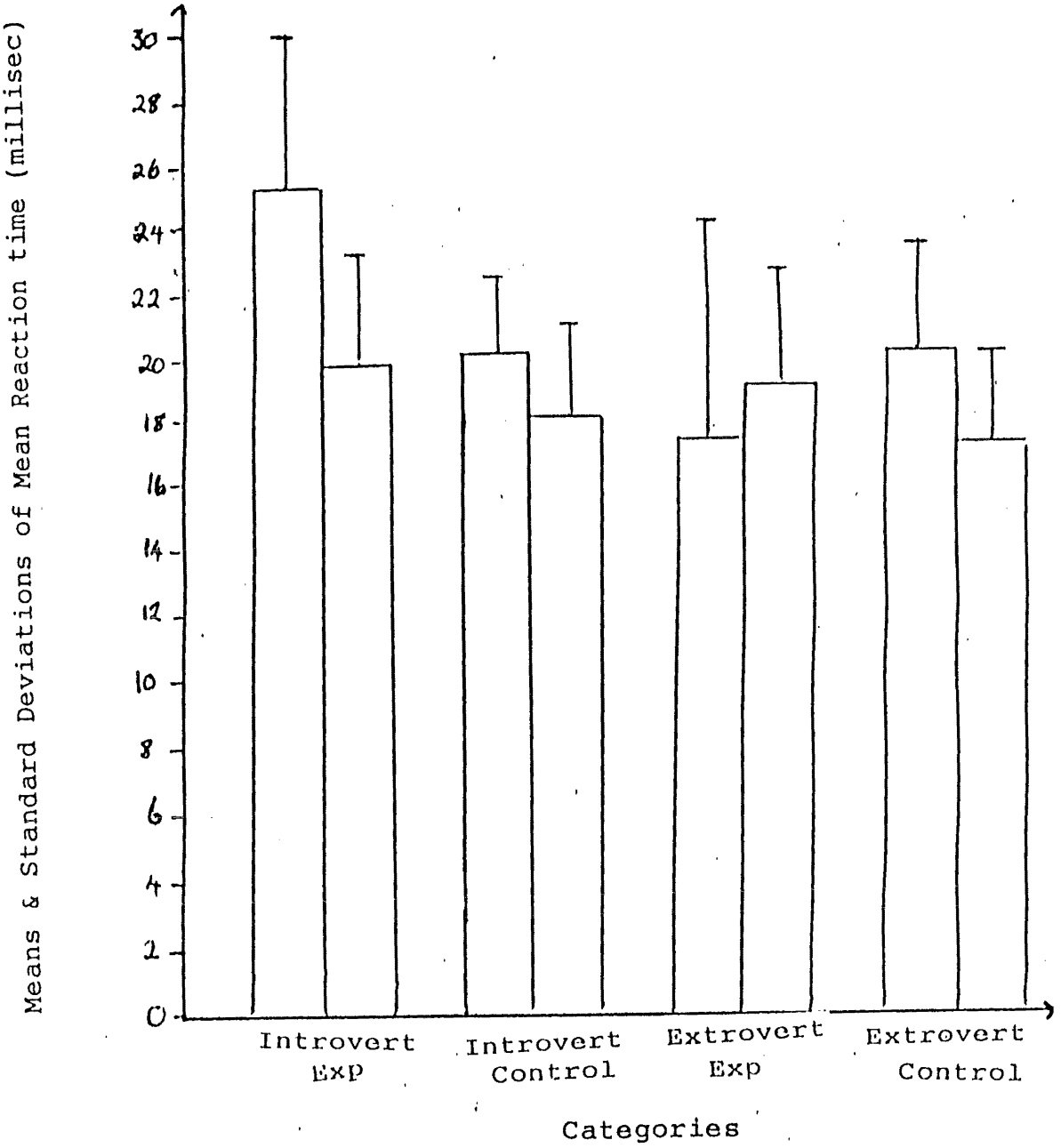


Figure (25)

The Means and Standard Deviations of Mood 1 (Relaxed/Restless) at the two Pre-treatment levels for each separate category.

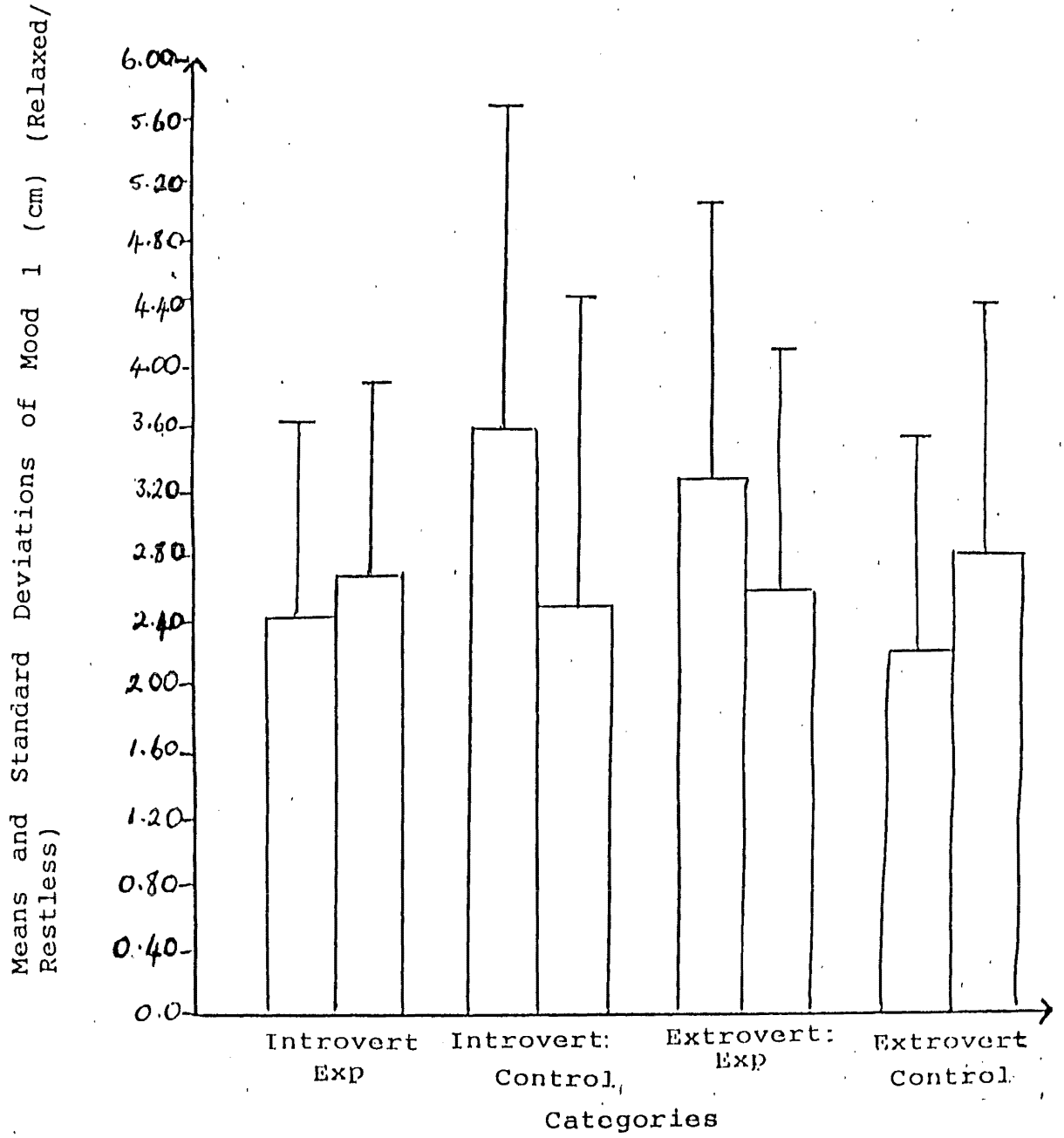


Figure 26. The means and standard deviations (normal breathing/breathlessness) at the 2 pre-treatment levels for each separate category.

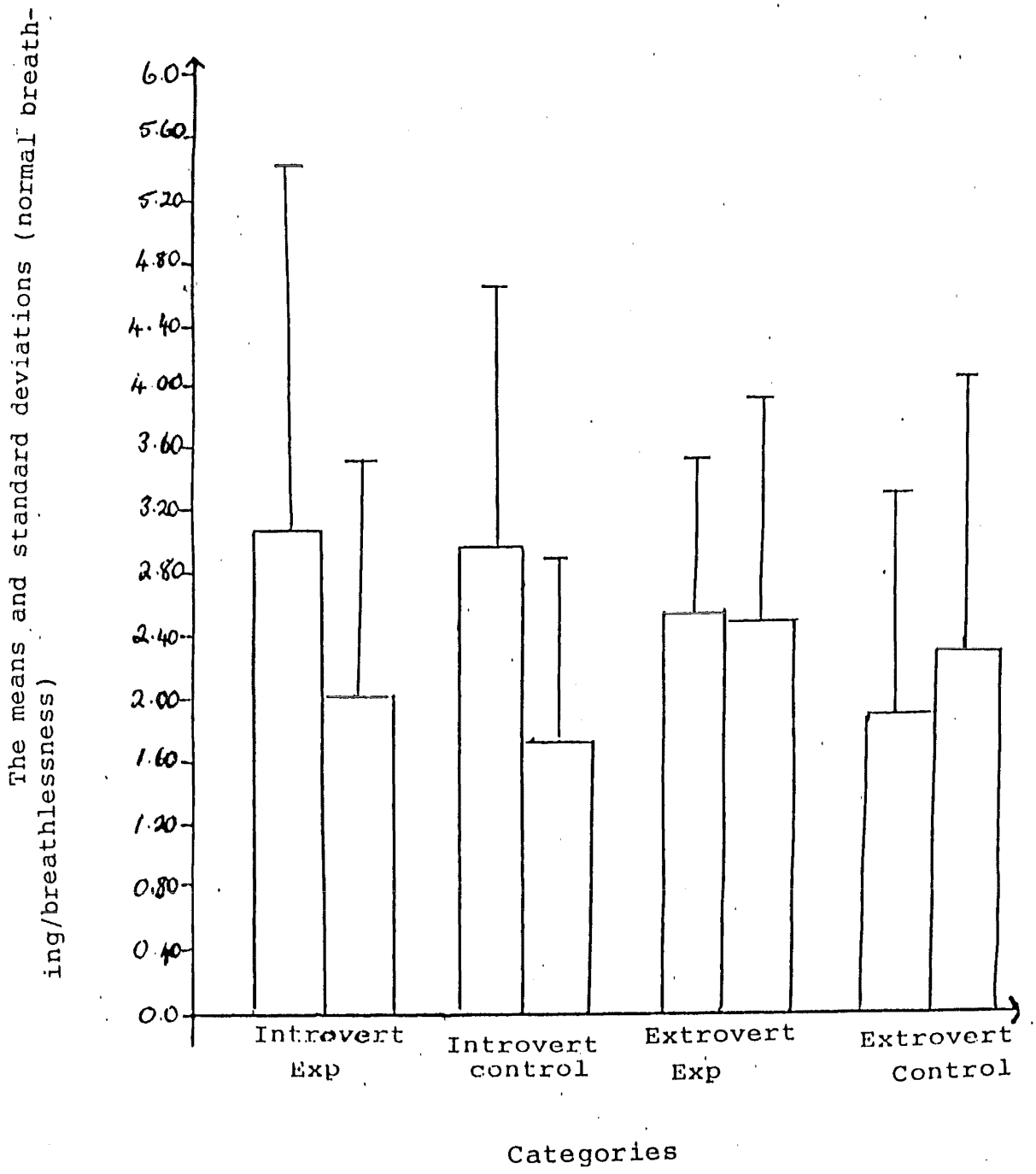


Figure (27)

The Means and Standard Deviations of Mood 3 (Sleepy/Alert) at the two Pre-treatment levels for each separate category.

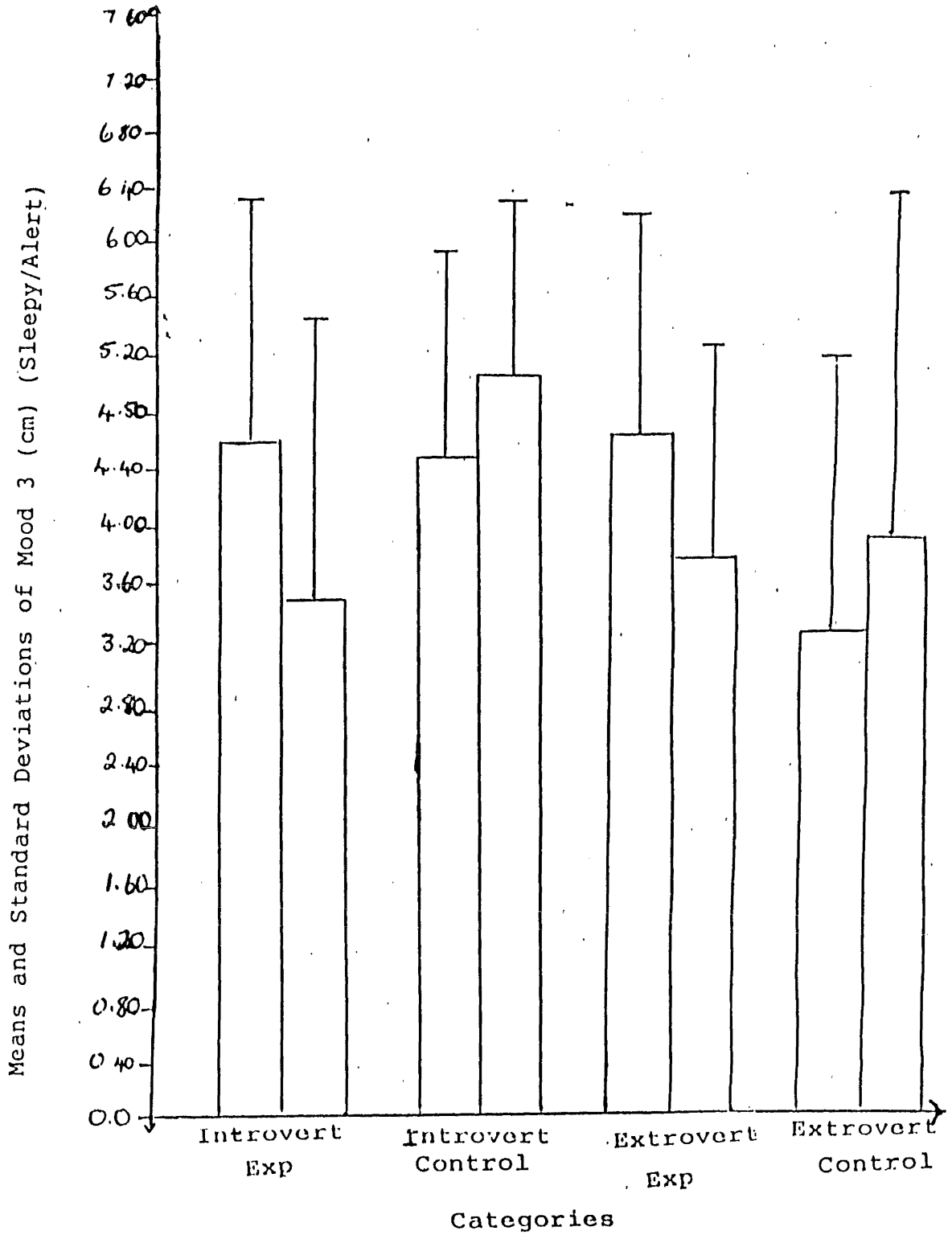


Figure (28)

The Means and Standard Deviations of Mood 4 (Unhappy/happy) at the two Pre-treatment levels for each separate category.

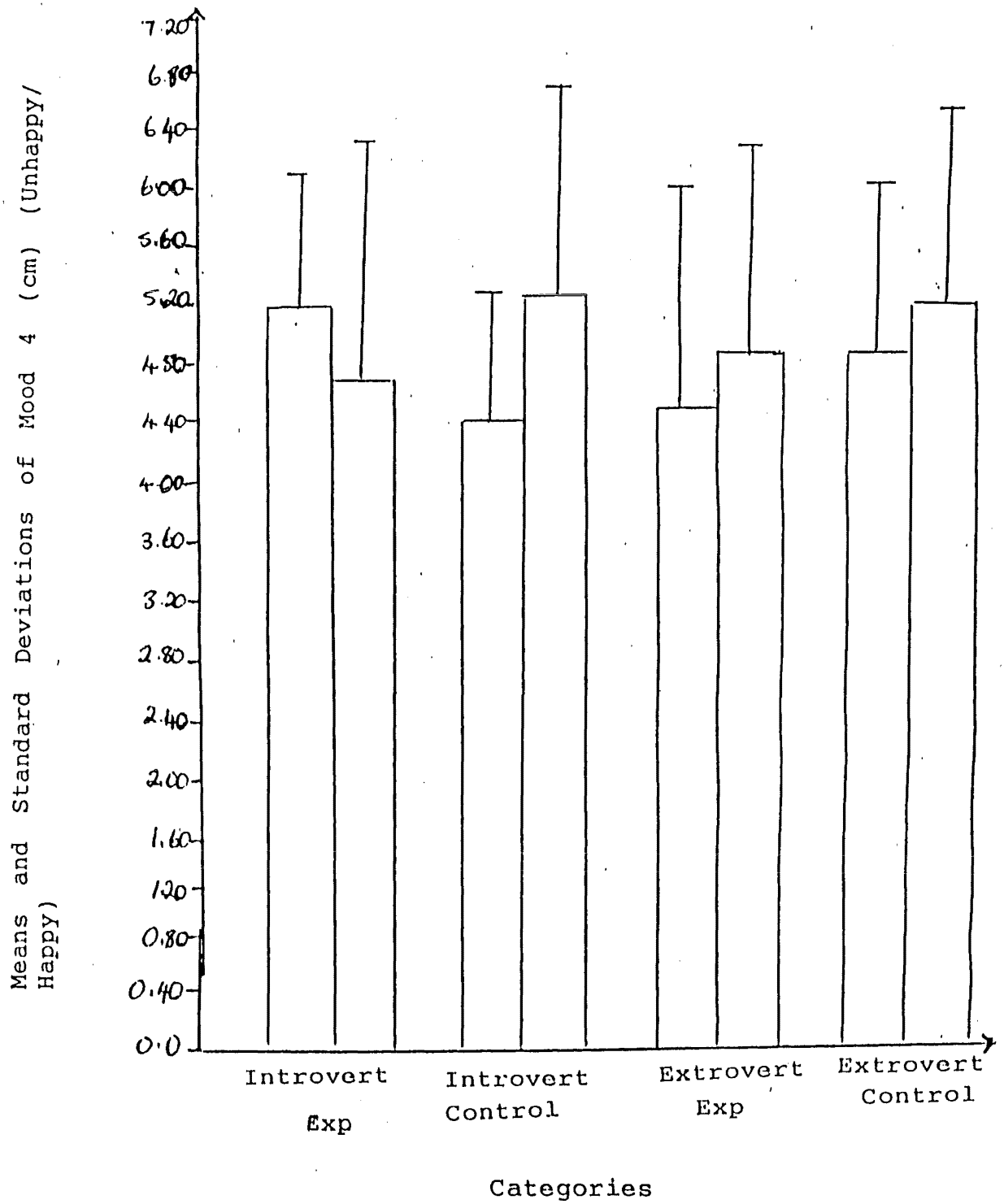


Figure (29)

The Means and Standard Deviations of Mood 5 (Calm/nervous) at the two Pre-treatment levels for each separate category.

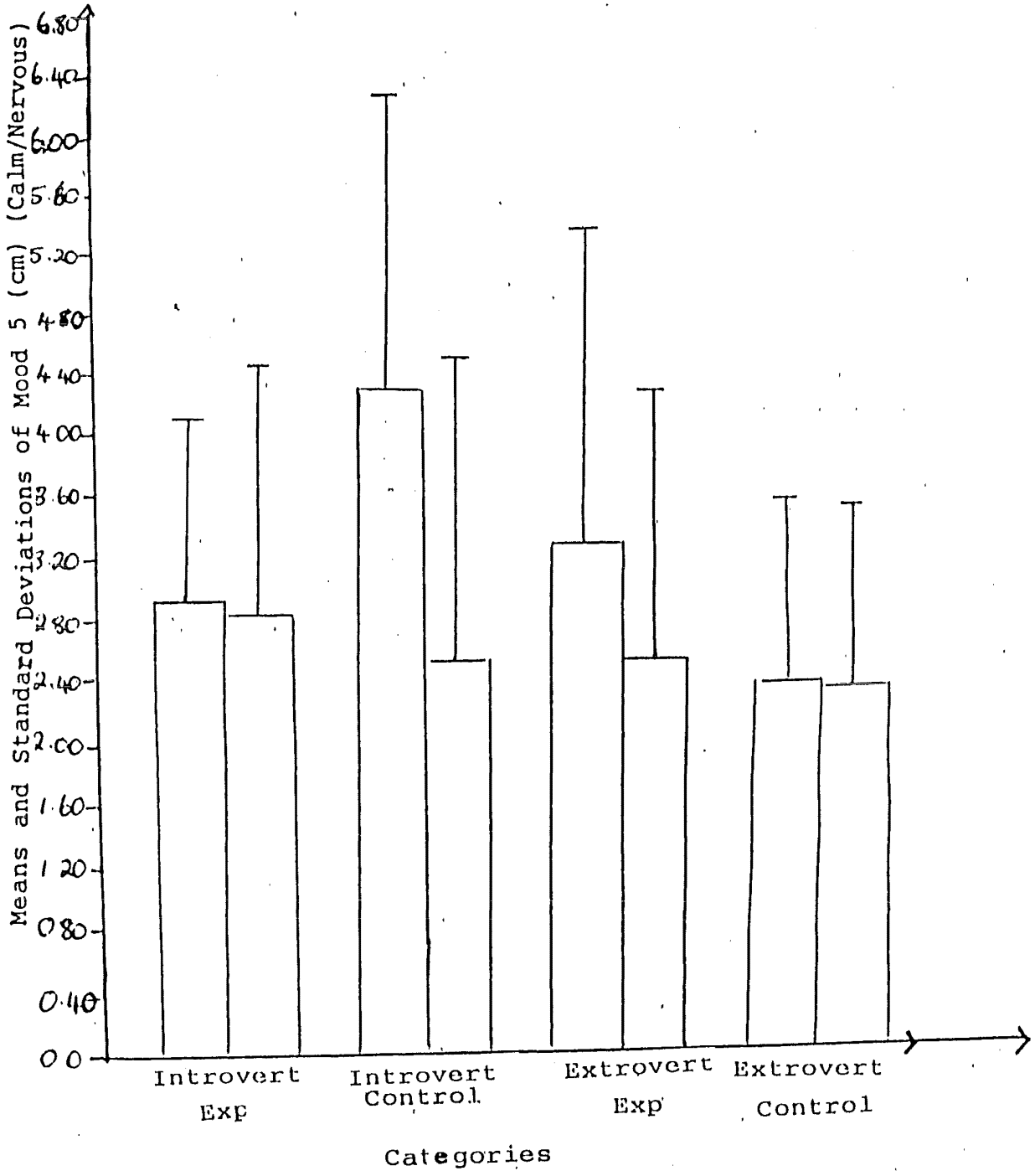


Figure (30)

The Means and Standar.. Deviations of Mood 6 (Confidence lacking/confident) at the two Pre-treatment levels for each separate category.

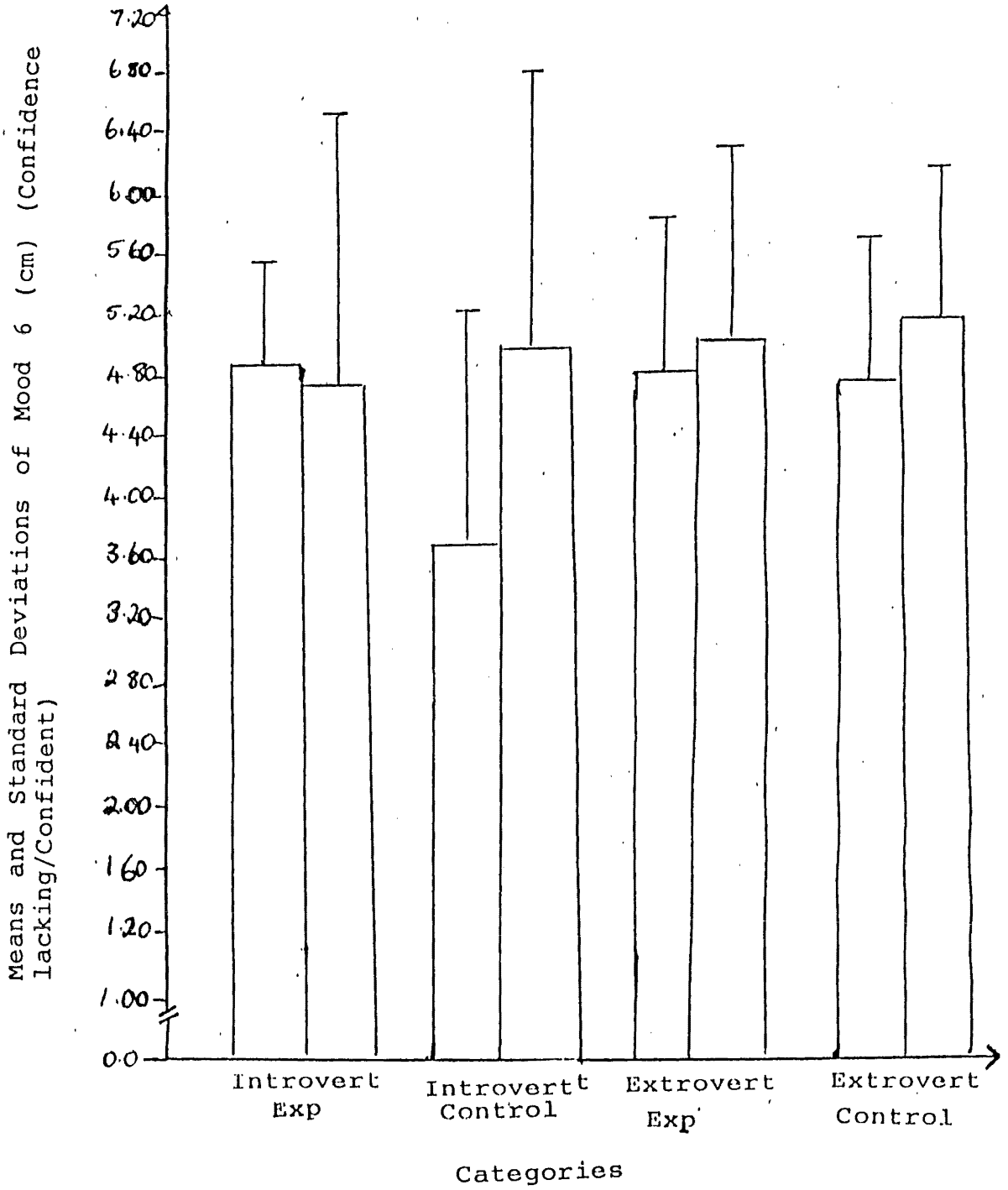


Figure (31)

The Means and Standard Deviations of Systolic Blood Pressure at the two Pre-Post treatment levels for each separate category.

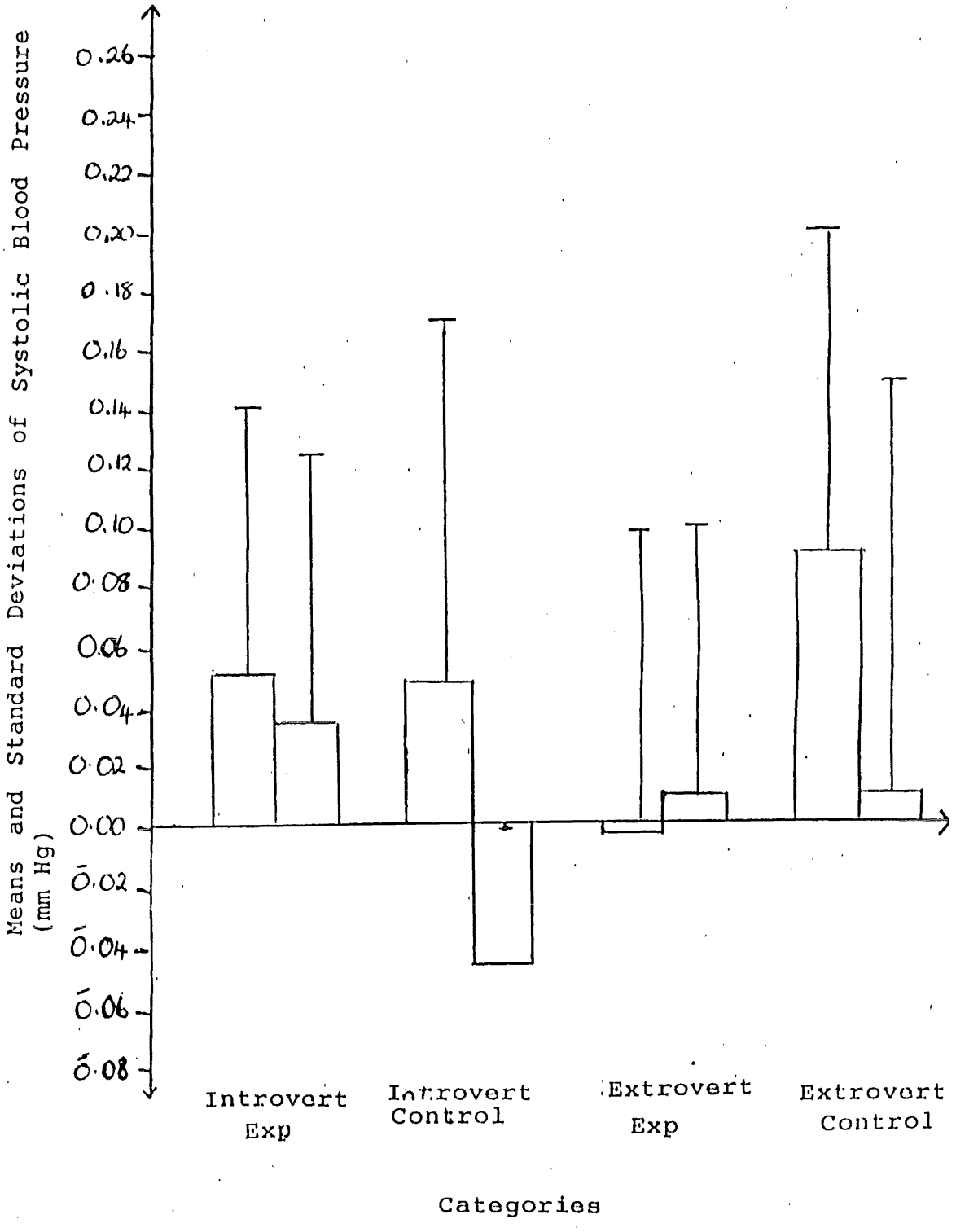


Figure (32)

The Means and Standard Deviations of Diastolic Blood Pressure at the two Pre-Post treatment levels for each separate category.

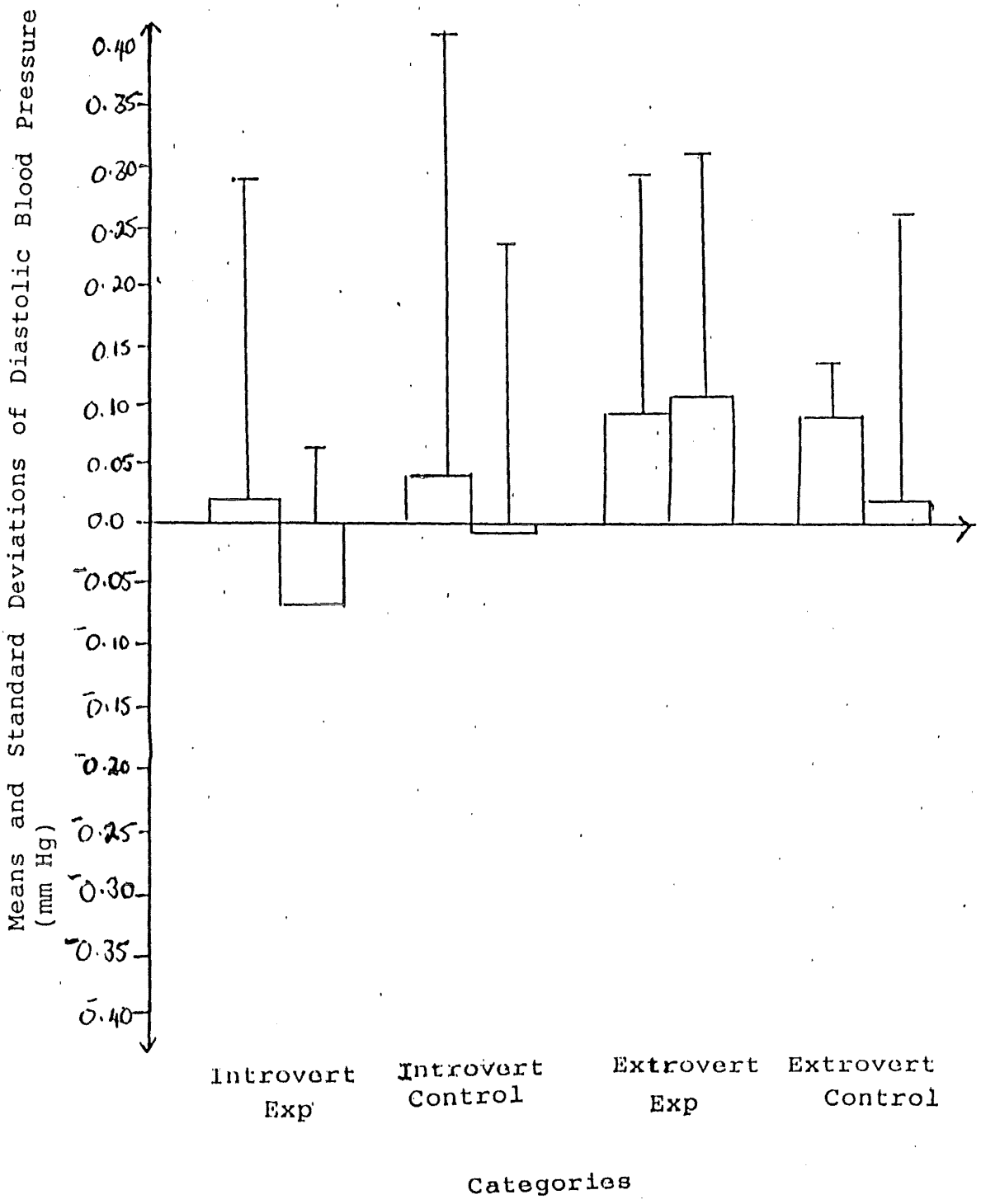


Figure (33)

The Means and Standard Deviations of Pulse Rate at the two Pre-Post treatment levels for each separate category.

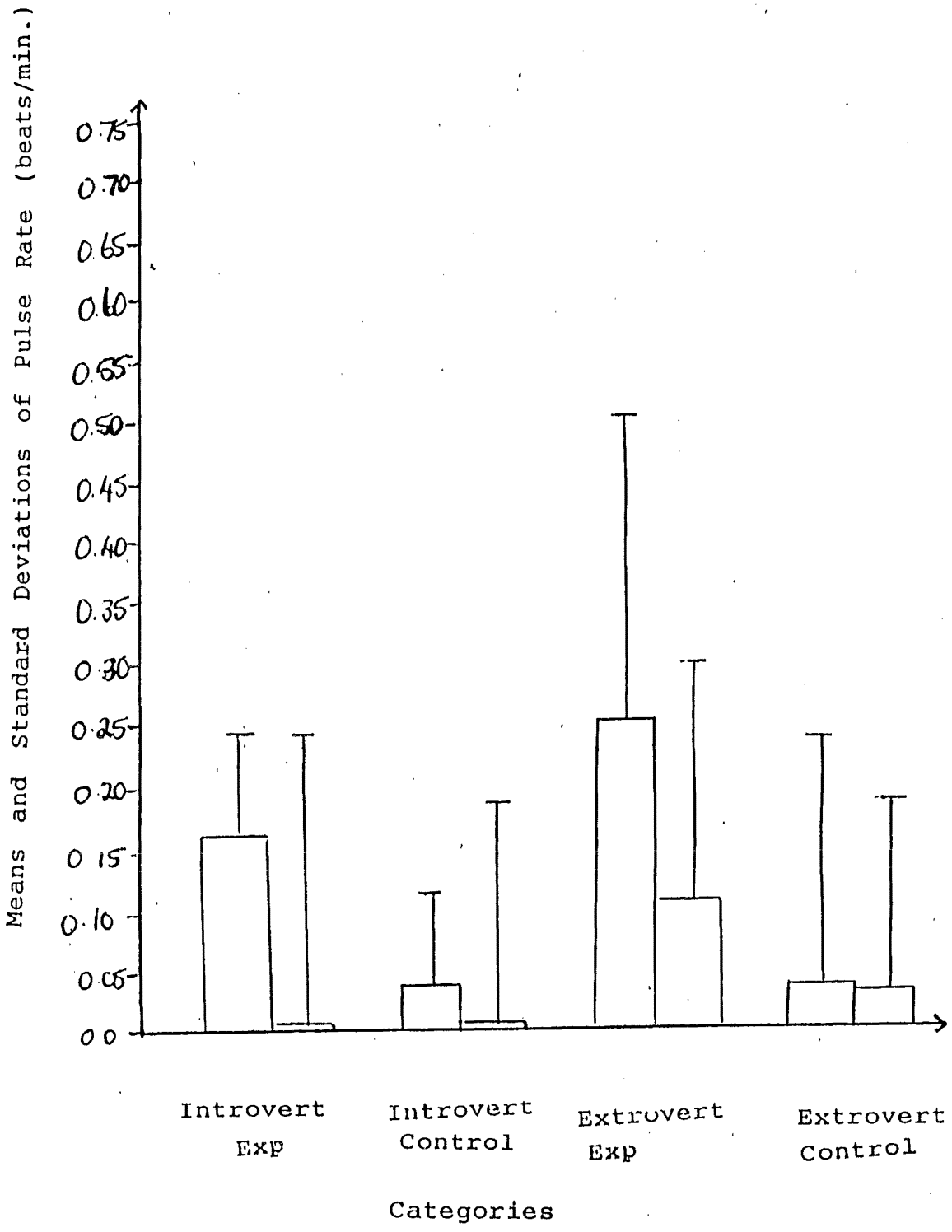


Figure (34)

The Means and Standard Deviations of Mean Reaction times at the Pre-Post treatment levels for each separate category.

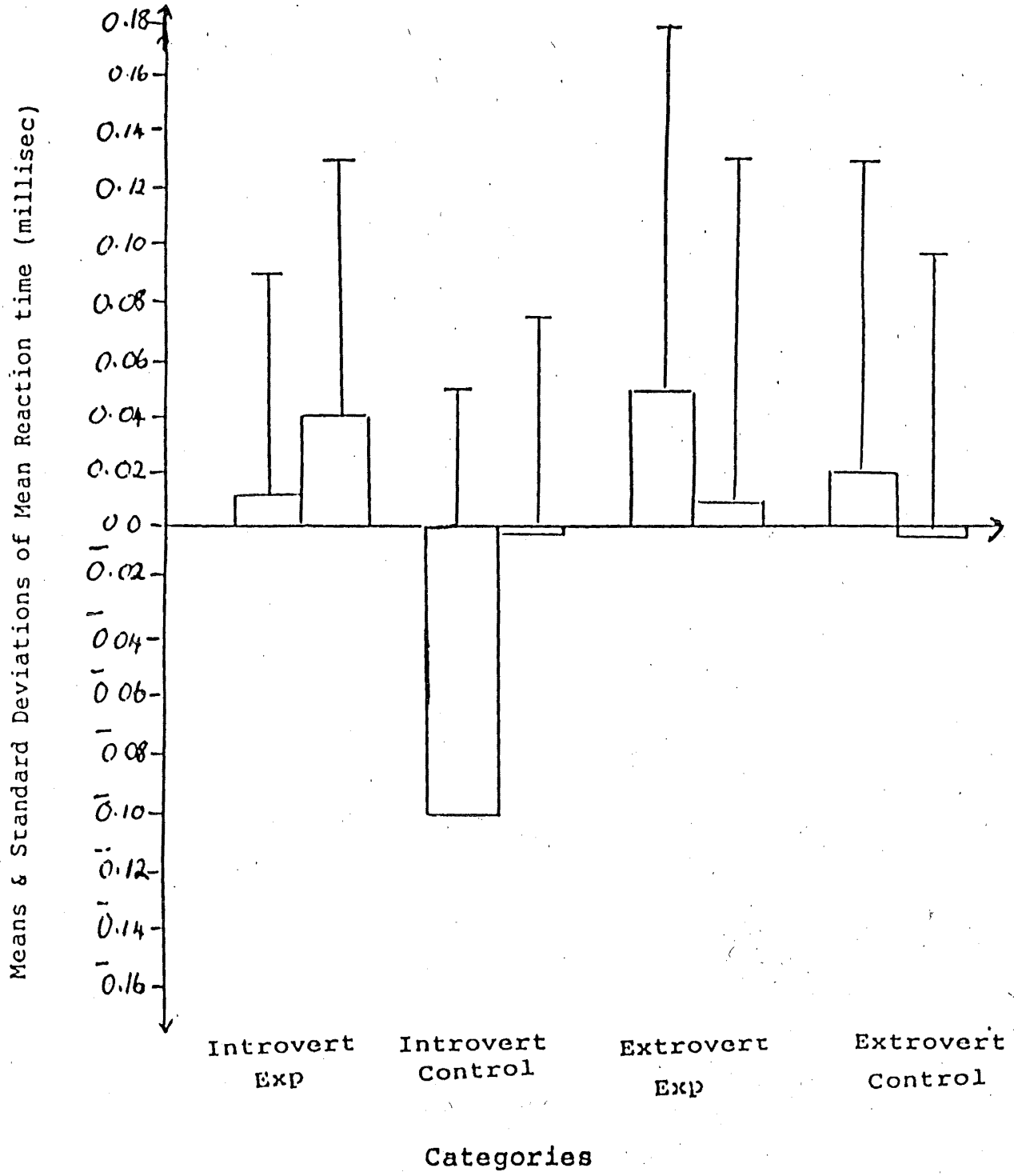


Figure (35)

The Means and Standard Deviations of Mood 1 (Relaxed/Restless) at the two Pre-Post treatment levels for each separate category.

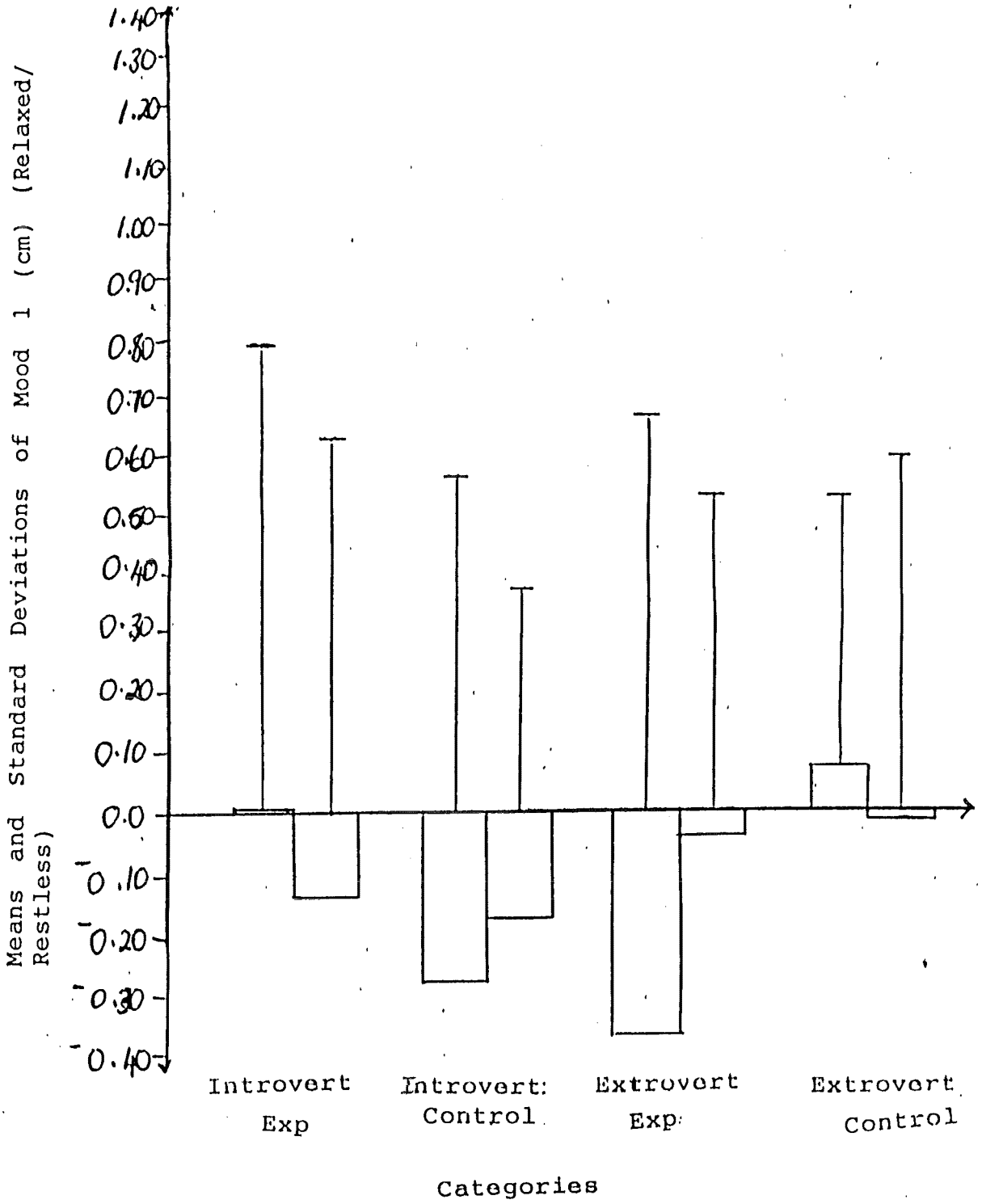


Figure 36.

The means and standard deviations (normal breathing/breathlessness) at the 2 pre-post treatment levels for each separate category.

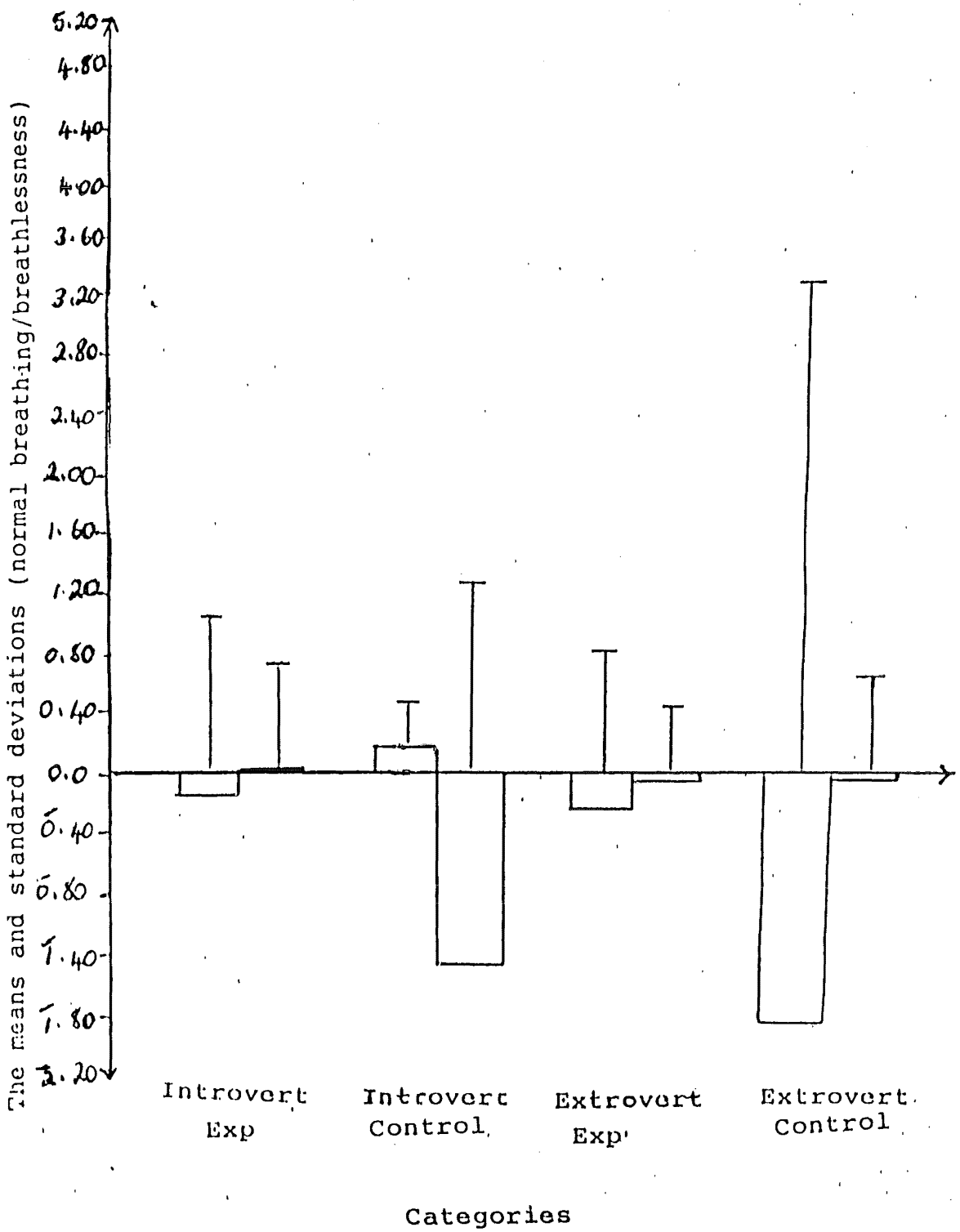


Figure (37)

The Means and Standard Deviations of Mood 3 (Sleepy/Alert) at the two Pre-Post treatment levels for each separate category.

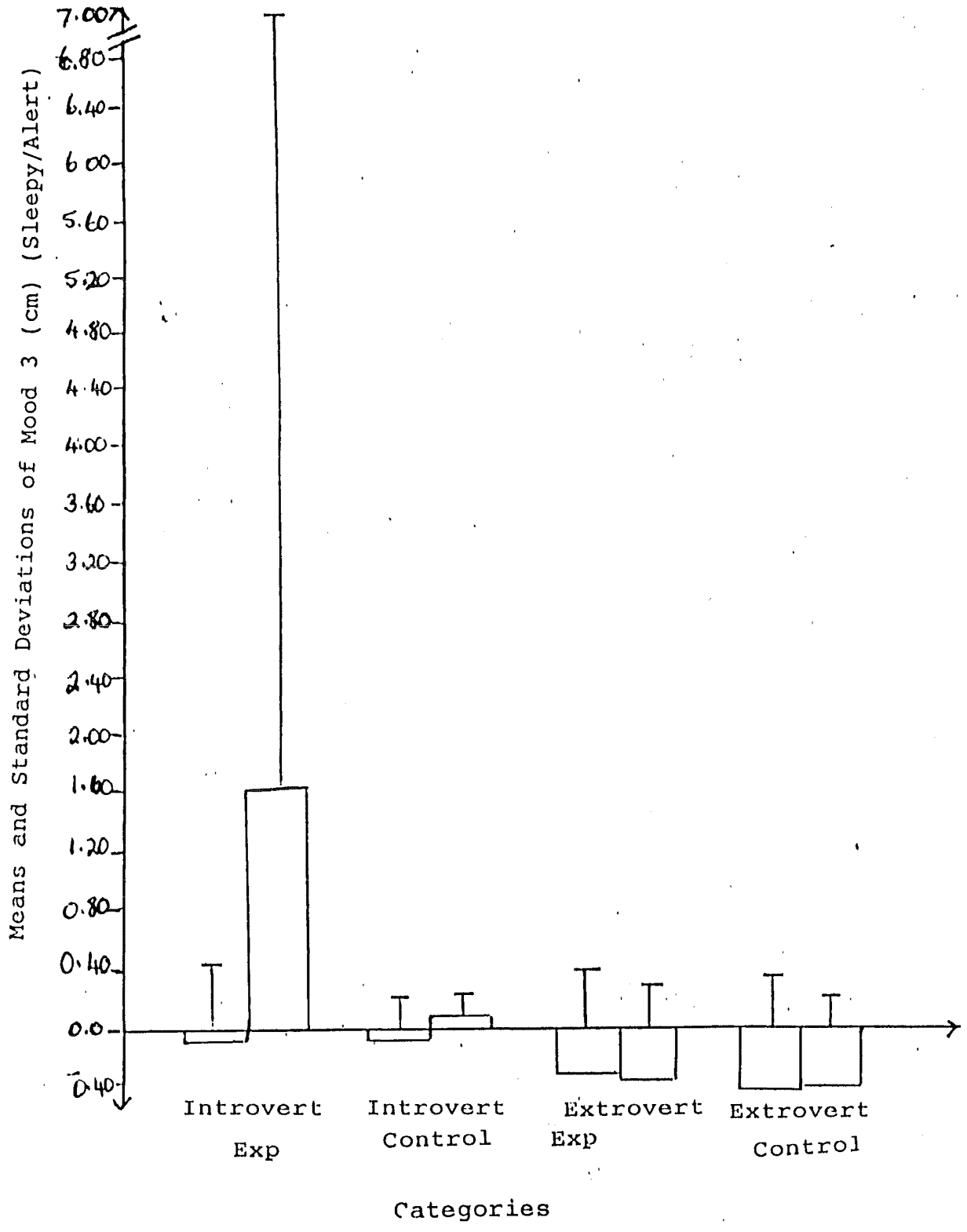


Figure (38)

The Means and Standard Deviations of Mood 4 (Unhappy/happy) at the two Pre-Post treatment levels for each separate category.

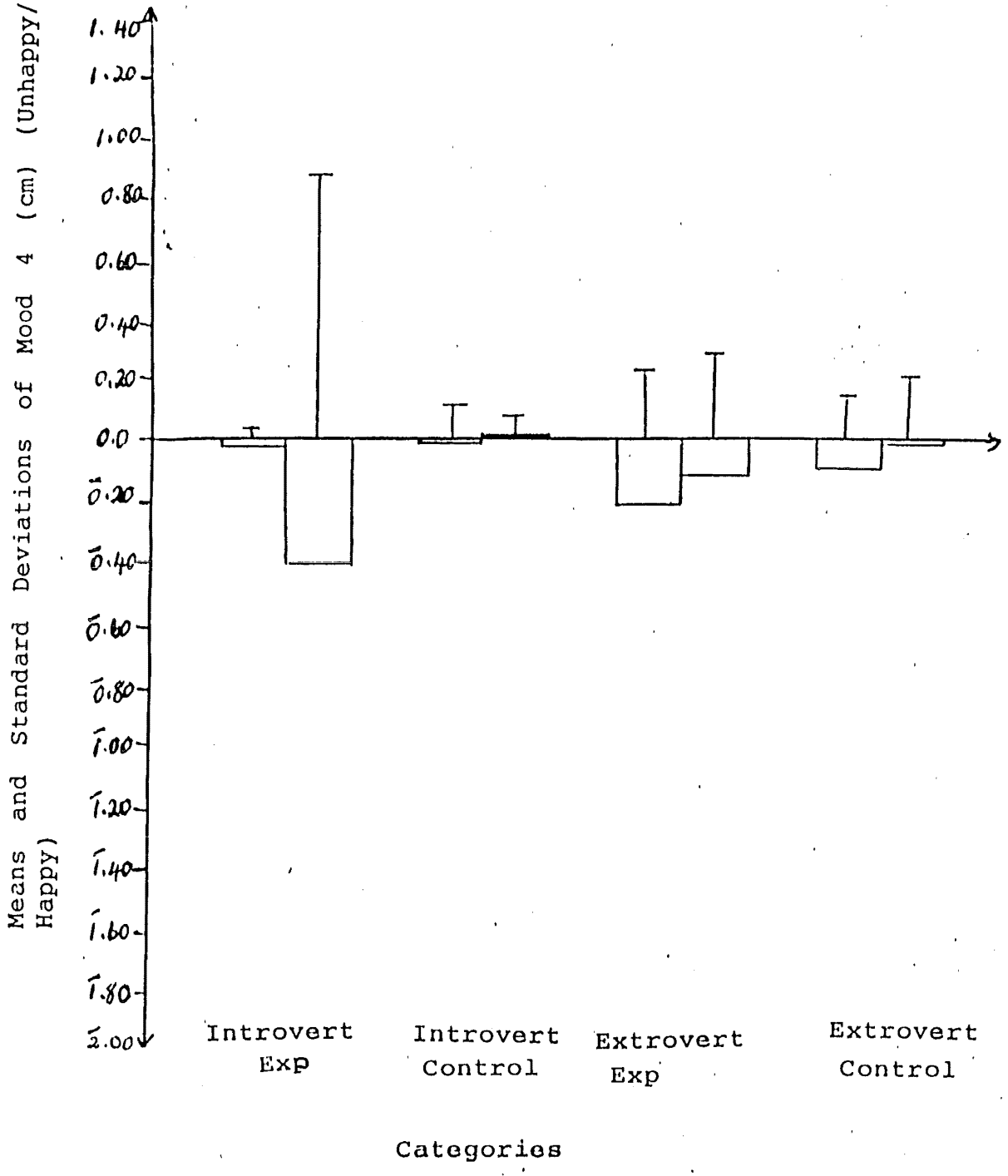


Figure (39)

The Means and Standard Deviations of Mood 5 (Calm/Nervous) at the two Pre-Post treatment levels for each separate category.

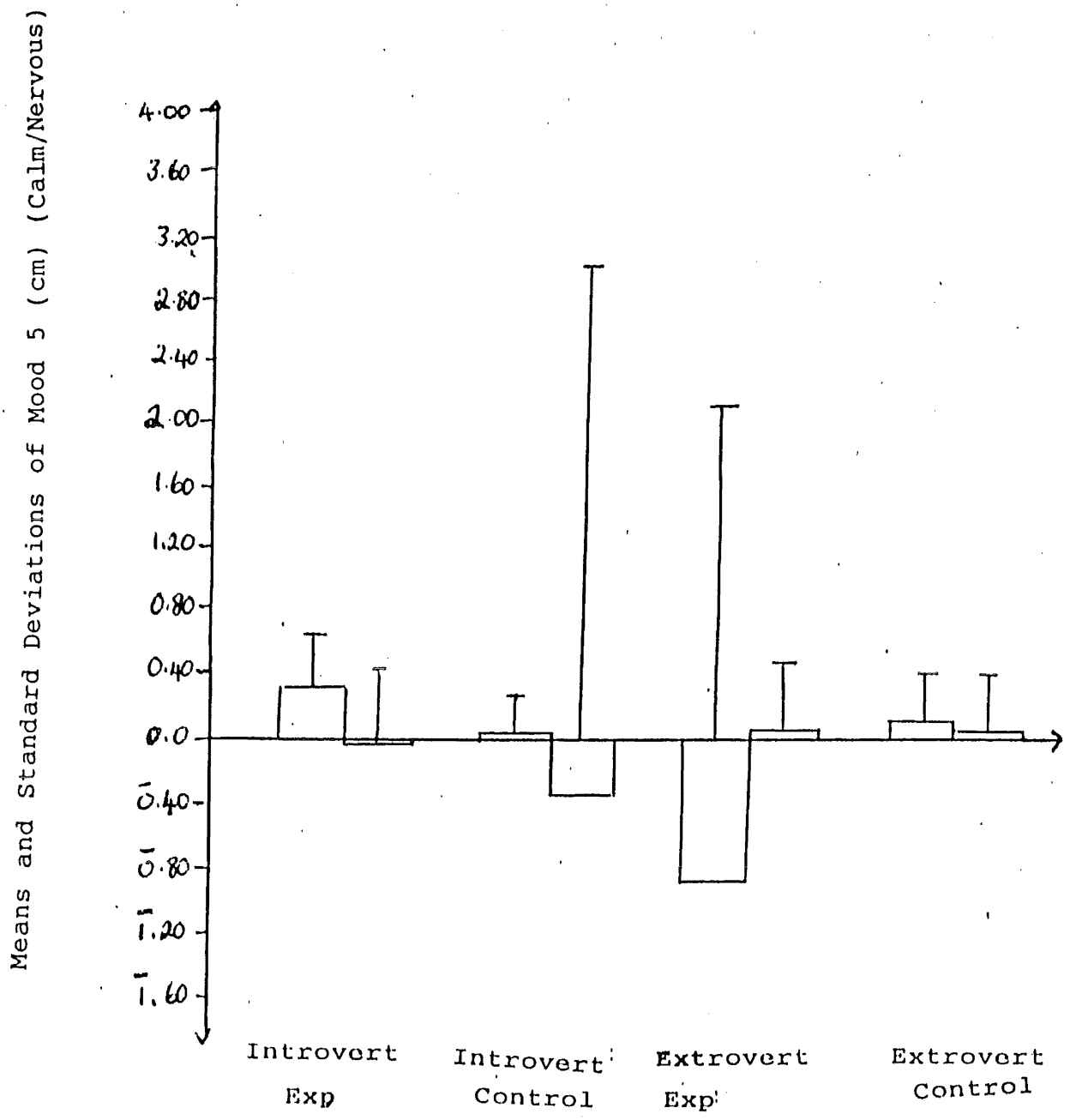
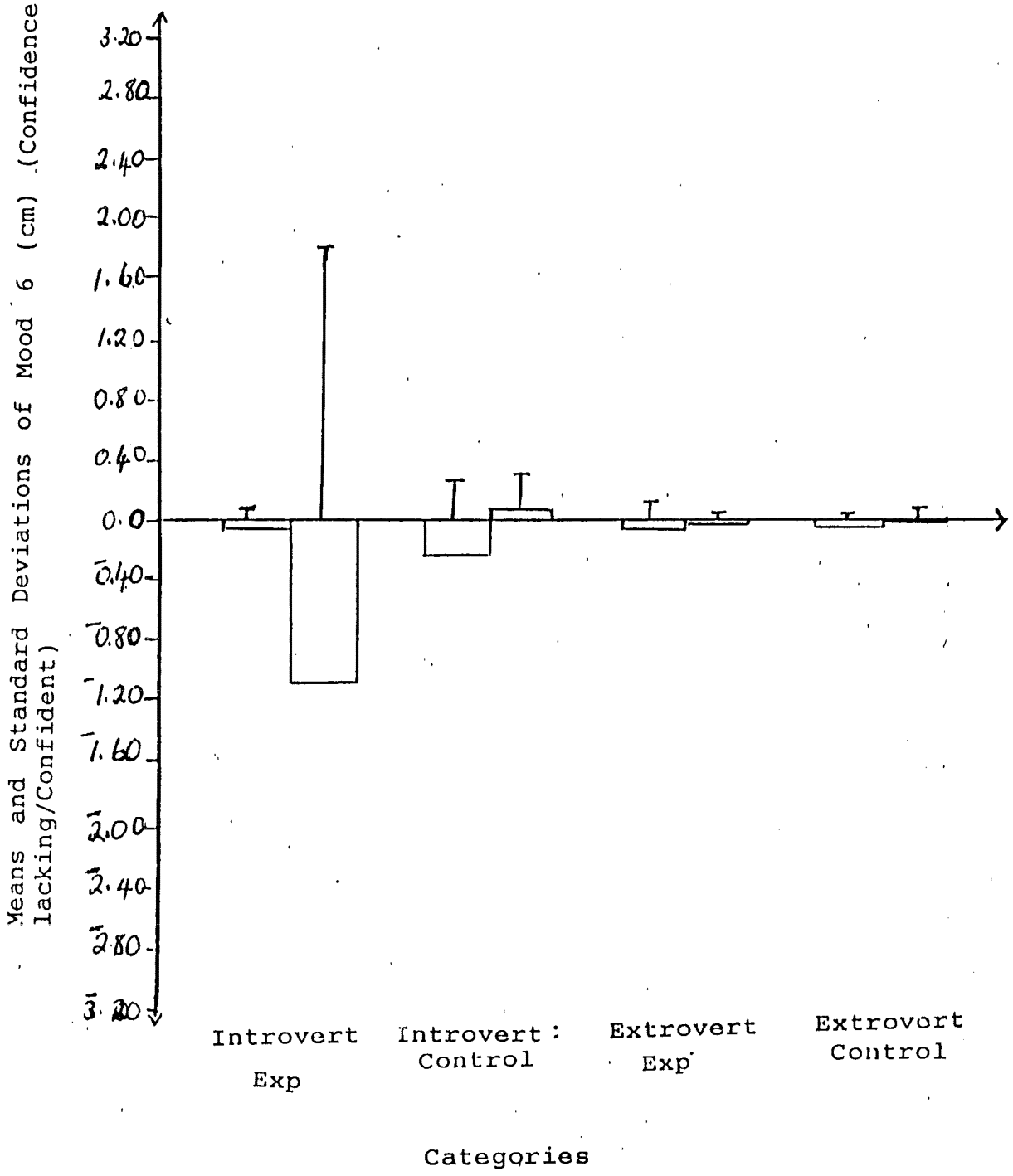


Figure (40)

The Means and Standard Deviations of Mood 6 (Confidence lacking/confident) at the two Pre-Post treatment levels for each separate category



CHAPTER VII

DISCUSSION

The general rationale adopted in this research was to test the validity of the Yerkes-Dodson Curvulinear Theory of Arousal (1908) and subsequently the validity of the Eysenck Personality Theory of Arousal using a simple reaction time task. Under this theory, it was expected that differences in responding (with respect to mean reaction time, blood pressure, pulse and mood) between introverts and extroverts would occur and would be accentuated by caffeine. Introverts were expected to demonstrate higher levels of arousal than extroverts post-caffeine and to have a lower level of optimal arousal when compared with extroverts.

The reaction time task was used because it is a useful parameter in the investigation of caffeine action and because findings of previous research provided a means of comparison for this st dy.

1. ANALYSIS ONE

With respect to the physiological parameters in analysis one, males tended to demonstrate higher systolic and diastolic blood pressures in the pre-caffeine phase than females and there was a systematic decrease in these two measures of blood pressure over the three sessions.

(See figures 1 and 2.) Previous research carried out by Cecil (1985) documented that males have higher blood pressures than females by the fourth decade which lends support to this observation. Statistically significant trends were found as a result of blood pressure measurements and although they give us insight into the Yerkes-Dodson Curvulinear Theory of Arousal, the magnitude of these trends was so small that in clinical settings, they could not be reliably used as predictive measures.

The systematic downward trend in the pre-caffeine systolic blood pressure phase of analysis one was not replicated in the pre-post difference phase. (See figure 11). A significant sessions effect occurred in the latter indicating an increase in systolic blood pressures after caffeine in the initial difference phase followed by decrements in blood pressure in the second difference phase and increases in systolic blood pressure in the third and final difference phase after caffeine. This trend was not followed by extrovert females, however, their differences in systolic blood pressure were much smaller by comparison. These results indicate that arousal was greatest on the initial and third difference phases if measured by systolic blood pressure difference. (It would have been interesting to demonstrate differences in arousal between the personality groups at still larger doses. However, ethical limitations prevented this.)

The results of figure (12) with respect to pre-post diastolic blood pressure difference indicate that at the

lowest caffeine dose, the highest diastolic blood pressure post caffeine was yielded. Males yielded higher diastolic blood pressure differences after caffeine than females on all three sessions. Personality did not appear to play any role in the pre-post difference phases for either of systolic or diastolic blood pressure.

Pulse rate did not yield any significant effects in either the pre-caffeine or the pre-post caffeine difference phase. This result was not totally unexpected as previous research using pulse rate as a parameter (Horst & Jenkins, 1934; Horst et al., 1934) had also depicted varying pulse rates with no obvious trend in either direction. With respect to this research, the variability in pulse rate can not be directly attributed to any of the variables under investigation. However, it is suggested that if larger cell sizes had been used, a trend may have been observed, as was the case discussed in Starr et al. (1937).

The electronic sphygmamanometer was used in all instances to obtain acute blood pressure and heart rate measures. Unfortunately, on several occasions, sudden movements by subjects resulted in changes in blood pressure and heart rate measures and these occurrences yielded error readings. When this occurred, a maximum of three attempts were made to obtain these measures. This decision was made because it is often clinically observed that if an excessive number of readings are repeatedly taken, measures of blood pressure and heart rate become lower

than normal. On the several occasions during which accuracy was diminished, an average blood pressure and pulse rate were determined on the basis of the individuals' two remaining sessions. This technique was instigated so as to maintain cell sizes.

As has been documented elsewhere, caffeine has the propensity to reduce reaction times. Consequently, the task is to determine if there are any attributes within individuals which determine the amount of change in reaction time. Eysenck stated that the reaction time for introverts is expected to decrease when caffeine is administered until optimum performance ensues. At this point, performance diminishes due to overarousal, while extroverts are expected to sustain and maintain optimal levels of performance at higher caffeine doses.

The reaction time scores in this research were an average of the twenty trials from each presentation of the stimulus. A significant personality x sessions x sex interaction effect occurred in the pre-caffeine phase of analysis one. The mean reaction times of introverts were higher (poorer performance) than those of the extroverts while females demonstrated greater consistency than males over the three sessions. By way of contrast, the pre-post mean reaction time difference phase yielded a significant sessions x personality interaction effect. Introverts yielded longer mean reaction times in both the initial and third difference phases than in the second difference phase. That is to say that they performed at their

optimum level at the second difference phase where average caffeine dose was approximately 100 mg. The third phase was characterised by overarousal and decrements in mean reaction time performance ensued. (See figure 14).

Extroverts demonstrated very rapid mean reaction times post caffeine on the initial difference phase, when compared with pre-caffeine, hence the negative measurement. However on the second and third difference phase reaction time post-caffeine was longer than the first. These results imply that the optimal reaction times for extroverts in this research occurred at 79 mg of caffeine.

These mean reaction time results lend partial support for the Eysenck Personality Theory in that the introverts demonstrated the proposed arousal pattern. This can not be said of the extroverts, however, who according to figure (14) optimized performance at the lowest caffeine level and produced the poorest performance at the highest dose (approximately 209 mg). This is contrary to the theory of arousal and consequently directly questions its validity.

However, this trend by the extroverts with respect to pre-post difference in mean reaction time may plausibly be explained in terms of the ethical constraints restricting higher doses of caffeine. Although the third difference phase depicted longer mean reaction time, it is probable that reaction time scores would be enhanced at the post caffeine phase if sufficient caffeine was administered. As was mentioned in the Methods section, the

extremities of introversion-extroversion on the Eysenck scale were used for subject selection, and consequently larger doses of caffeine would be required to produce readily observable improvements in performance in many of the extroverted participants.

A significant personality x sex interaction effect occurred in the pre-caffeine phase of mood 2 (Normal breathing/Breathlessness). Extrovert males and females demonstrated more breathlessness on the third session than on the first two while a reverse trend was demonstrated for introverts.

A significant personality x sex interaction effect occurred in the pre-post difference phase for mood 1 (Relaxed/Restless). The extroverts depicted more uniformity than the introverts over the three difference phases and a higher level of restlessness at the third difference phase, characterised by the highest caffeine dose. A significant sessions effect occurred for the pre-post difference phase for mood 2 (Normal breathing/Breathlessness). The initial and third sessions for each of the four groups yielded measures of breathlessness and extroverts demonstrated their highest level of breathlessness on the third difference phase which also characterised the highest caffeine dose. These are results which are expected, especially for extroverts who sustain optimal performance at higher levels of caffeine and restlessness and breathlessness are strong indicants of arousal.

2. ANALYSIS TWO

With respect to the physiological and psychological parameters of analysis two, results on the pre-dose systolic blood pressure phase depict higher systolic blood pressure among control group members than the experimental group and systolic blood pressure decreases on the second phase, whereas the opposite occurs for the experimental group. There was also a significant sessions effect for the pre-dose diastolic blood pressure. For each of the four groups under investigation, the second pre-dose diastolic blood pressure was lower than the first and this trend closely mirrors the pre-caffeine changes in the first analysis. The pre-post difference phase for systolic blood pressure depicted the largest difference between the two phases for introverted and extroverted control members. (See figure 31). The results depict lower post-dose systolic blood pressures than pre-dose for three of the four groups for the second stimulus, excluding the extroverted experimental group who depicted a positive trend from the initial phase to the second indicating that post-dose systolic blood pressure was higher than pre-dose for the second stimulus. It should be noted that the difference between the two difference phases was smallest for extroverted experimental group members.

Because the largest difference between the two phases belonged to the control group members, it is suggested that personality rather than caffeine has affected

this change as no caffeine was administered to the control group. Members of the introverted experimental group demonstrated a decrease between pre-post difference phases in which the second session (the stop sign) yielded a lower post-dose systolic blood pressure than in session one. The extroverted experimental group demonstrated the opposite effect, in which initial measurement yielded a larger pre than post measure, however the post-dose systolic blood pressure was larger than the pre-dose measure for the second session.

It would have been interesting to compare the pre-dose and pre-post dose differences of analysis two with the corresponding measure in analysis one. However, this type of comparison was prohibited because the variable Exp/Control was substituted for sex on analysis two.

This occurred because the predictive value of a four-way analysis of variance, using sex which had not yielded many significant effects, would not have been enhanced by interpretation of four-way interaction effects for a research population of this size.

There was a significant pre-dose effect on diastolic blood pressure in analysis two. Figure (22) depicts the systematic decrease in diastolic blood pressure from the initial session to the second session. The difference between the two sessions was most obvious for those members of the control group, while the differences between the two sessions for the experimental group in the pre-dose phase were considerably less. It appears that

members of the control group have an attribute or numerous attributes which combine to produce greater variability in systolic and diastolic blood pressure. Possibilities include the way in which they respond to stress and anxiety, as it is a well-established fact that anxiety and discomfort or other stress can induce transient increases in blood pressure.

Figure (24) depicts a significant personality x sessions x Exp/Control interaction effect in which a lower pre-dose mean reaction time was obtained in the second session when compared to the first in each of the four groups, excluding the extroverted experimental group in which the opposite trend occurred. A possible explanation of this phenomenon is that the second session was perceived as less threatening or stressful than the initial session by the participants and consequently reaction time decreased. Results from figures (22) and (23) with respect to systolic and diastolic blood pressures demonstrate trends in the same direction as the trend for mean reaction time, adding weight to the hypothesised explanation. These three graphs all demonstrate the opposite trend for the extroverted experimental group which indicates that they perform better under stress (see figure 24) than introverts.

However, when pre-post mean reaction time phases were considered, personality was no longer important as was the case in analysis one (figure 14) and the experiment/control variable became significant. The results

indicated that rather than personality determining responses on the two sessions, caffeine administration appeared to be more important. (See figure 33). It is conceivable that although a personality effect may be present, the caffeine dose may be masking this smaller trend by its presence. Figure (33) demonstrates large initial session post-dose mean reaction times among the experimental group when compared to the initial session for the control group members. They also characterise the largest change between sessions one and two with second session measures for the experimental group indicating lower post-dose mean reaction times than in session one. It is interesting that differences occur between the two sessions of the magnitude demonstrated by the experimental group and it appears that the caffeine administration has enhanced the variability of responding in the experimental group. An alternative explanation could be that members of the experimental group are more susceptible to practice effects than members of the control group. However, there is no information in the literature supporting the concept of practice effects and perhaps more research should ensue in this area.

A consideration is that at this point in the research, the experimental group have already undergone three trials as compared with one for control group members and this familiarity could in fact contribute significantly to a proposed practice effect. It should also be considered that mean reaction time is measured on

figure (33) by increments of 0.05 milisecs, which are very small units. Although statistical significance is maintained, it is questionable as to the clinical relevance of such small changes. It may be that considerably larger doses of caffeine have to be administered before large differences in mean reaction time on this task are observed.

The final results of significance under discussion are pre-dose mood variables, the first of which relates to breathing. Figure (26) demonstrated a larger difference between the two pre-dose sessions for introverts, than was evident for extroverts. Introverts demonstrated more breathlessness on the initial session than on the second (the flashing stop sign). This is an interesting result because there was no significant effect in analysis one for this mood variable. It is conceivable that introverts responded with less enthusiasm on the second session than extroverts because they perceived the simplicity of the task as demanding of less arousal while the extroverts maintained the opposite perception.

Mood 3 (sleepy/alert) showed a significant sessions x Exp/Control interaction effect as is demonstrated in figure (27). The experimental group showed larger decreases in alertness between the two pre-dose sessions than the control group members. A possible reason for this is that they had undergone numerous sessions prior to analysis two commencing unlike control group members. Consequently the task had relatively less novelty for them

than it held for the control group and required less attention.

3. CONCLUDING COMMENTS

Controversy still reigns as to the predictive qualities of the Introversion/Extroversion dimension. While this research was not intended as a critical appraisal, it has offered data which questions the validity of this dimension. It has also incorporated the Yerkes-Dodson Curvulinear Theory of Arousal, to provide a systematic investigation into the wide variety of effects promoted by caffeine.

The first objective of this research was to identify the differences between the response systems of two distinct personality groups with respect to mean reaction time, blood pressure, heart rate and mood. A second objective was to examine the extent to which these differences would be accentuated through the action of caffeine. As has been documented in the preceding sections, there was limited uniformity in the results obtained from both analysis one and analysis two.

The pre-caffeine measures of mean reaction time for analysis one imply a poorer performance by introverts than extroverts, for stimuli of the same strength. This result tends to support Eysenck's hypothesis of different patterns of physiological arousal for the two personality types which produce differences in optimal levels.

However, the expectation was that introverts having higher levels of arousal than extroverts, would have demonstrated better pre-caffeine mean reaction times than extroverts. This was not the case. The pre-post difference phase for mean reaction time on analysis one also depicted unusual results. Optimal performance for introverts occurred at 100 mgs of caffeine while optimal mean reaction time for extroverts occurred at a considerably lower dose of 75 mgs of caffeine. The pre-post mean reaction time measures for analysis two failed to yield any significant personality effects. This raises the question as to whether these findings were subject to a masking effect by caffeine.

The findings of this research do not lend support to Eysenck's Personality Theory using the introversion/extroversion dimension and can not be fully explained by the Yerkes-Dodson Curvulinear Theory of Arousal. This is not proposed as an alternative to Eysenck's Personality Theory but rather as a part of that theory because the inconsistencies can be partially explained in terms of subject sensitivity. It is not inconceivable that in order for behavioural and cognitive effects of caffeine use to become evident, an individual may have to demonstrate a heightened level of sensitivity to caffeine. This may be even more apparent in research such as this which makes use of small cell sizes (N = 34). These and the personality differences may be due to the already recognised differences in neuronal systems with respect to adenosine activity. It is this aspect of arousal which is

worthy of further investigation in association with the Eysenck Personality Theory and the Yerkes-Dodson Curvulinear Theory of Arousal in order to further an understanding of the interplay between arousal and personality types and the subsequent outcome on physiological and psychological parameters.

Acknowledgements

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References

Alstott, R.L., Miller, A.J., & Forney, R.B. (1973). Report on human fatality due to caffeine. Forensic Sciences, 18(12), pp.135-137.

Anderson, K.J. & Revelle, W. (1983). The interactive effects of caffeine, impulsivity and task demands on a visual search task. Personality and Individual Differences, 4(2), pp.127-134.

Axelrod, J. & Reichenthal, J. (1954). The fate of caffeine in man and a method for its estimation in biological material. Journal of Pharmacology and Experimental Therapeutics, 107, pp.519-523.

Boulenger, J.P., Patel, J., Post, R.M., Parma, A.M., & Marangos, P.J. (1983). Chronic caffeine consumption increases the number of brain adenosine receptors. Life Sciences, 32, pp.1135-1142.

Bowyer, P.A., Humphreys, M.S. & Revelle, W. (1983). Arousal and recognition memory: The effects of impulsivity, caffeine and time on task. Personality and Individual Differences, 4, pp.41-49.

Broadbent, D. (1958). Perception and Communication. London: Pergamon Press.

Bross, L.D.J. & Tidings, J. (1973). Another look at coffee drinking and cancer of the urinary bladder. Preventive Medicine, 2, pp.445-451.

Carpenter, J.A. (1959). The effect of caffeine and alcohol on simple visual reaction time. Journal of Comparative and Physiological Psychology, 52, pp.491-496.

Carpenter, J.A. (1962). Effects of alcohol on some psychological process. Quarterly Journal of Studies on Alcohol, 23, pp.274-314.

Carrrigan, P.M. (1960). Extroversion-Introversion as a dimension of personality: A reappraisal. Psychological Bulletin, 57, pp.329-360.

Cecil Textbook of Medicine 17th edition. (1985). Wyngaarden & Smith, W.B. Saunders & Co., U.S.A.

Charney, D.S., Galloway, M.P., & Heninger, G.R. (1984). The effects of caffeine on plasma MHPG, subjective anxiety, autonomic symptoms in blood pressure in healthy humans. Life Sciences, 35, pp.135-144.

Charney, D.S., Heninger, G.R., & Jatlow, P.L. (1985). Increased anxiogenic effects of caffeine in panic disorders. Archives of General Psychiatry, 42(3), pp.233-243.

Cheney, R.H. (1935). Comparative effect of caffeine per se and a caffeine beverage upon reaction time in normal young adults. Journal of Pharmacology and Experimental Therapeutics, 53, pp.304-313.

Cherek, D.R., Steinberg, J.L., & Brauchi, J.T. (1983). Effects of caffeine on human aggressive behaviour. Psychiatry Research, 8, pp.137-145.

Cherek, D.R., Steinberg, M.L., & Brauchi, J.T. (1984). Regular or decaffeinated coffee and subsequent human aggressive behaviour. Psychiatry Research, 11(3), pp.251-258.

Chou, D.T., Khan, S., Forde, J., & Hush, K.R. (1985). Caffeine tolerance: Behavioural, electrophysiological and neurochemical evidence. Life Sciences, 36, pp.2347-2358.

Cole, P. (1971). Coffee drinking and cancer of the lower urinary tract. Lancet, 1, pp.1335-1337.

Dalby, J.T. (1985). Will population decreases in caffeine consumption unveil attention deficit disorders in adults? Medical Hypotheses, 18, pp.163-167.

Daly, J.W., Burns, R.F., & Snyder, S.H. (1981). Life Sciences, 28, pp.2083-2097.

Dimaio, V.J.M. & Garriott, J.C. (1974). Lethal caffeine poisoning in a child. Forensic Science, 3, pp.275-278.

Downing, R.W. & Rickels, K. (1981). Coffee consumption, cigarette smoking and reporting of drowsiness in anxious patients treated with benzodiazepines or placebo. Acta Psychiatrica Scandinavica, 64, pp.398-408.

Dreisback, R.H. & Pfeiffer, C. (1943). Caffeine withdrawal headache. Journal of Laboratory Clinical Medicine, 28, pp.1212-1219.

Easterbrook, J.A. (1959). The effects of emotion on cue utilization and the organization of behaviour. Psychological Review, 66, pp.183-201.

Elsass, P. (1986). Continuous reaction time in cerebral dysfunction. Acta Neurologica Scandinavia, 73, pp.225-246.

Erikson, G.C., Hager, Houseworth, Dugan, Petros & Beckwith (1985). The effects of caffeine on memory for word lists. Physiology and Behaviour, 35(1), pp.47-51.

Eysenck, H.J. (ed.). (1963a). Experiments With Drugs. Oxford: Pergamon Press.

Eysenck, H.J. (ed.). (1963b). Experiments in Motivation. Oxford: Pergamon Press.

Eysenck, H.J. (1967). The Biological Basis of Personality. Illinois: Thomas Springfield.

Eysenck, M.W. (1976). Arousal, learning and memory. Psychological Bulletin, 83, pp.390-404.

Eysenck, M.W. & Folkard, S. (1980). Personality, time of day and caffeine: Some theoretical and conceptual problems. In Revelle et al. Journal of Experimental Psychology General, 10?, pp.32-41.

Eysenck, H.J. & Levy, A. (1972). Conditioning Introversion/Extraversion and the strength of the nervous system. In V.D. Nebylitsyn & J.A. Gray (eds) Biological Bases of Individual Behaviour. New York: Academic Press.

Firestone, P. Poitras-Wright, H., & Douglas, V. (1978). The effects of caffeine on hyperactive children. Journal of Learning Disabilities, 11, pp.133.

Freedman, A.M., Kaplan, I.I. & Saddock, B.J. (eds). (1975). Modern synopsis of comprehensive textbook of psychiatry. Baltimore: Williams & Wilkins Co., 2nd edition.

Freedman, A.M., Kaplan, I.I. & Saddock, B.J. (eds). (1978). Modern synopsis of comprehensive textbook of psychiatry. Baltimore: Williams & Wilkins Co., 3rd edition,

Freedman, A.M., Kaplan, I.I. & Saddock, B.J. (eds). (1981). Modern synopsis of comprehensive textbook of psychiatry. Baltimore: Williams & Wilkins Co., 4th edition.

de Freitas, B. & Schwartz, G. (1979). Effects of caffeine in chronic psychiatric patients. American Journal of Psychiatry, 136(10), pp.1337-1338.

Garfinkel, B.D., Webster, C.D., & Solman, L. (1981). Responses to methlphenidate and varied doses of caffeine in children with attention deficit disorder. Canadian Journal of Psychiatry, 26(6), pp.395-401.

Gilbert, R.M. (1981). Caffeine. Overview and Anthology in Nutrition and Behaviour. Philadelphia, PA: Franklin Institute Press, pp.145-166.

Gilliland, K. (1980). The interactive effect of introversion-extroversion with caffeine induced arousal on verbal performance. Journal of Research on Personality, 14, pp.482-492.

Graham, D.M. (1978). Caffeine its identity, dietary sources, intake and biological effects. Nutrition Research (Review), 36, pp.97-102.

Greden, J.F. (1974). Anxiety or caffeinism: A diagnostic dilemma. American Journal of Psychiatry, 131, pp.1089-1092.

Greden, J.F., Lee Myung Ae & Cameron, O.G. (1985). Anxiety and caffeine consumption in people with anxiety disorders. Psychiatry Research, 15(3), pp.211-217.

Greden, J.F. Fontaine, P., Lubetsky, M., & Chamberlain, K. (1978). Anxiety and depression associated with caffeinism among psychiatric inpatients. American Journal of Psychiatry, 135, pp.963-966.

Greden, J.F. Victor, B., Fontaine, P., & Lubetsky, M. (1980). Caffeine withdrawal headache: A clinical profile. Psychosomatics, 21, pp.411-418.

Green, R.M. & Stiles, G.L. (1986). Chronic caffeine ingestion sensitizes the A1 adenosine receptor-adenylate cycalse system in rat cerebral cortex. Journal of Clinical Investigation, 77, pp.222-227.

Grollman, A. (1930). The action of alcohol, caffeine and tobacco on the cardiac output (and its related functions) of normal man. Journal of Pharmacology and Experimental Therapeutics, 39, pp.313-327.

Guilford, J.P. (1975). Factors and factors of personality. Psychological Bulletin, 82, pp.802-814.

Gilliland, K. & Andress, D. (1981). Ad lib caffeine consumption symptoms of caffeinism and academic performance. American Journal of Psychiatry, 138, pp.512-514.

Gilliland, K. & Bullock, W. (1983/1984). Caffeine: A potential drug of abuse. Advances in Alcohol and Substance Abuse, 3(1/2), pp.53-73.

Goldstein, A., Warren, R. & Kaiser, S. (1965a). Psychotropic effects of caffeine in man I. individual differences in sensitivity to caffeine induced wake fulness. Journal of Pharmacology and Experimental Therapeutics, 14, pp.156-159.

Goldstein, A., Kaiser, S., & Warren, R. (1965b). Psychotropic effects of caffeine in man II. Alertness, psychomotor coordination and mood. Journal of Pharmacology and Experimental Therapeutics, 15, pp.146-151.

Goldstein, A. Kaiser, S., & Whitby, O. (1969). Psychotropic effects of caffeine in man IV. Quantitative and qualitative differences associated with habituation to coffee. Clinical Pharmacological Therapeutics, 10, pp.489-497.

Gong, H., Simmons, M.S., Tashkin, D.P., Hui, K.K., & Lee, E.Y. (1986). Bronchodilator effects of caffeine in coffee. A dose response study of asthmatic subjects. Chest, 89(3), pp.335-342.

Goodman, A. & Gilman, . (eds) (1980). Pharmacological Basis of Therapeutics, 6th ed. N.Y.: Macmillan.

Guilford, J.P. (1977). Will the real factor of extroversion-introversion please stand up: A reply to Eysenck. Psychological Bulletin, 84, pp.412-416.

Gupta, S. & Nicholson, J. (1985). Simple visual reaction time, personality and strength of the nervous system: A signal detection theory approach. Personal Individual Differences, 6(4), pp. 61-469.

Hamilton, P., Hockey, G.R. & Quinn, J.G. (1972). Information selection, arousal and memory. British Journal of Psychology, 63(2), pp.181-189.

Harvey, D.H. & Marsh, R.W. (1978). The effects of decaffeinated coffee versus whole coffee on hyperactive children. Developmental and Medical Child Neurology, 20, pp.81-86.

Hayre, S.S. (1973). Coffee drinking and acute myocardial infarction. Lancet, 1, p.45.

Hockey, G.R., Davis, S. & Gray, M.M. (1972). Forgetting as a function of sleep at different times of day. Quarterly Journal of F perimental Psychology, 24(4), pp.386-393.

Hollingsworth, H.L. (1912). The influence of caffeine on mental and motor deficiency. Archives of Psychology, 3, pp.1-16.

Horst, K. & Jenkins, W.L. (1935). The effect of caffeine, coffee and decaffeinated coffee upon blood pressure, pulse rate and simple reaction time of men of various ages. Journal of Pharmacology, 53, pp.385-400.

Horst, K., Robinson, W.D., Jenkins, W.L., & Bao, D.L. (1934). The effects of caffeine, coffee and decaffeinated coffee upon blood pressure, pulse rate and certain motor reactions of normal young men. Journal of Pharmacology and Experimental Therapeutics, 52(3), pp.

Jacobs, M.H., Senior, R.M., & Kessler, A. (1976). Clinical experience with theophylline: Relationships between dosage serum concentration and toxicity. Journal of the American Medical Association, 235, pp.1983-1986.

Kordash, T.R., Van Dallen, R.G., & McCall, J.T. (1977). Theophylline concentrations in asthmatic patients after administration of aminophylline. Journal of the American Medical Association, 238, pp.139-141.

Lee, M.A., Cameron, O.G., & Greden, J.F. (1985). Anxiety and caffeine consumption in people with anxiety disorders. Psychiatry Research, 15, pp.211-217.

Lutz, E.G. (1978). Restless legs, anxiety and caffeinism. Journal of Clinical Psychiatry, 39(9), pp.693-698.

MacMahon, B., Yen, S., Trichopoulos, D., Warren, K. & Nardi, G. (1981). Coffee and cancer of the pancreas. New England Journal of Medicine, 304, pp.630-633.

Marangos, P.J., Boulenger, J.P., & Patel, J. (1984). Effects of chronic caffeine on brain adenosine receptors: Regional and antogenetic studies. Life Sciences, 34, pp.899-907.

Maren, T.H. (1961). The additive renal effect of oral aminophylline and trichlormethiazide in man. Clinical Research, 9, p.57.

Marks, V. & Kelly, J.K. (1973). Absorption of caffeine from tea, coffee and coca-cola. Lancet, 7807, p.827.

Mathew, R.J. & Wilson, W.H. (1985). Caffeine induced changes in cerebral circulation. Stroke, 16(5), pp.814-817.

Mathew, R.J. & Wilson, W.H. (1985). Caffeine consumption withdrawal and cerebral blood flow. Headache, 25, pp.305-309.

Murray, T. (1982). Up-regulation of rat cortical adenosine receptors following chronic administration of theophylline. European Journal of Pharmacology, 82, pp.113-114.

Myung, L.A. Cameron, O.G. & Greden, J.F. (1985). Anxiety and caffeine consumption in people with anxiety disorders. Psychiatry Research, 15(3), pp.211-217.

Nash, H. (1962). Alcohol and caffeine - A study of their physiological effects. New York: Raven Press.

Nash, H. (1966). Psychological effects and alcohol antagonizing properties of caffeine. Quarterly Journal of Studies in Alcohol, 27, pp.727-734.

Ogilvie, R.L., Fernandez, P.G. & Winsberg, F. (1977). Cardiovascular response to increasing theophylline concentrations. European Journal of Clinical Pharmacology, 12, pp.409-414.

Osborne, D.J. & Rogers, Y. (1983). Interactions of alcohol and caffeine on human reaction time. Aviation, Space and Environmental Medicine, pp.528-534.

Payne, R.B. & Hauty, G.T. (1955). Factors affecting the endurance of psychomotor skill. Journal of Aviation Medicine, 25, pp.382-389.

Piafsky, K.M., Sitar, D.S., Rango, R.E. & Ogilvie, R.L. (1977). Theophylline kinetics in acute pulmonary edemic. Clinical Pharmacology and Therapeutics, 21, pp.310-316.

Pilcher, J.D. (1912). Alcohol and caffeine. A study of antagonism and synergism. Journal of Pharmacological Experimental Therapeutics, 3, pp.267-298.

Posniak, P.C. (1985). The carcinogenicity of caffeine and coffee. A review. Journal of the American Dietic Association, 85, pp.1127-1133.

Posner, J., Papatestas, A.E. Fagerstrom, R., Schwart, L., Saevitz, J., & Feinberg, M. Association of tumor differentiation with caffeine and coffee intake in women with breast cancer. Surgery, 100(3), pp.482-488.

Rall, T.W. (1980). Central nervous system stimulants: The Xanthines. In The Pharmacological Basis of Therapeutics, 6th ed. New York: Macmillan, pp.192-607.

Rapaport, J.L., Berg, C.J., Ismanel, D.R. Zhan, T.P., & Neims, A. (1984). Renal effects of coffee in children: Relationship between dietary choice and the effects of caffeine challenge. Archives of General Psychiatry, 41(11), pp.1073-1079.

Reichard, C.C. & Elder, S.T. (1978). The effects of caffeine on reaction time in hyperkinetic and normal children. American Journal of Psychiatry, 134, p.144.

Revelle, W., Amaral, P., & Turiff, . (1976). Introversion/extroversion, stress and caffeine. Effect on verbal performance. Science, 192, pp.149-150.

Revelle, W., Humphreys, M.S., Simon, L., & Gilliland, K. (1980). The interactive effect of personality, time of day and caffeine. A test of the arousal market. Journal of Experimental Psychology General, 109, pp.1-31.

Ritchie, J.M. (1970). Central nervous system stimulants. II. The Xanthines. In The Pharmacological Basis of Therapeutics, 4th ed. Edited by Goodman, L.S. & Culman, A. New York: Macmillan Publishing Co., pp.358-370.

Sattin, A. & Rall, T.W. (1970). The effect of adenosine and adenine nucleotides on the adenosine 3'5'-phosphate content of guinea pig cerebral cortex slices. Molecular Pharmacology, 6, pp.13-23.

Sawyer, D.A., Julia, H.L. & Turin, A.C. (1982). Caffeine and human behaviour, arousal, anxiety and performance effects. Journal of Behavioural Medicine, 5, pp.415-439.

Schilling, W. (1921). The effect of caffeine and acetanilid on simple reaction time. Psychological Review, 28, pp.72-79.

Schnackenberg, R. (1973). Caffeine as a substitute for schedule two stimulants in hyperkinetic children. American Journal of Psychiatry, 130, pp.796-798.

Snyder, S.H. & Sklar, P. (1984). Psychiatric Progress: Behavioural and molecular actions of caffeine: Focus on adenosine. Journal of Psychiatric Research, 18(2), pp.91-106.

Soloman, R.L. & Corbit, J.D. (1974). An opponent-process theory of acquired motivation. Temporal dynamic of affect. Psychological Review, 81, pp.119-145.

Scriouphau, W.P.H. & Bracken, M.B. (1986). Caffeine consumption during pregnancy and association with late spontaneous abortion. American Journal of Obstetrics and Gynaecology, 154, pp.14-20.

Starr, L., Gamble, C.F., Marangolies, A. Donald, J.S., Joseph, N., & Eagle, E. (1937). A clinical study of the action of ten commonly used drugs on cardiac output, work and size, on respiration, on metabolic rate and on the electrocardiogram. Journal of Clinical Investigation, 16, pp.799-823.

Stocks, P. (1970). Cancer mortality in relation to national consumption of cigarettes, solid fuel, tea and coffee. British Journal of Cancer, 24, pp.215-225.

Sutherland, N.S., Carr, A.R., & Mackintosh, J.A. (1962). Visual discrimination of open and closed shapes by rats I. training II. transfer tests. Quarterly Journal of Experimental Psychology, 14(3), pp.129-156.

Svensson, E., Person, L. & Sgoberg, L. (1980). Mood effects of diazepam and caffeine. Psychopharmacology, 67(1), pp.73-80.

Thelle, D.S., Arnesen, E., & Forde, O.H. (1983). The Tromso heart study: Does caffeine raise serum cholesterol? New England Journal of Medicine, 38, pp.1454-1457.

Timson, J. (1977). Caffeine. Mutation Research, 47, pp.1-52.

Tokola, R.A. (1985). Tol enamic acid in combination with caffeine: Absorption during acute migraine. Methods and Findings in Experimental and Clinical Pharmacology, 7(10), pp.545-550.

Weathersbee, P.S., Olsen, L.K. & Lodge, J.R. (1977). Caffeine and pregnancy. Postgraduate Medicine, 62, pp.64-69.

Weiss, B. & Laites, V.A. (1962). Enhancement of human performance by caffeine and the amphetamines. Pharmacology Review, 14, pp.1-36.

Winstead, D.K. (1976). Coffee consumption among psychiatric inpatients. American Journal of Psychiatry, 133, pp.1447-1450.

Yerkes, R.M. & Dodson, J.D. (1908). The reaction of strength of stimuli to rapidity of habit formation. Journal of Comparative Neurology, 18, pp.459-482.

Appendix 1

E ○ N ○ L ○ ☐☐☐☐☐☐☐☐☐☐

FORM A

		YES	NO
1.	Do you often long for excitement?	○	○
2.	Do you often need understanding friei : to cheer you up?	○	○
3.	Are you usually carefree?	○	○
4.	Do you find it very hard to take no for an answer?	○	○
5.	Do you stop and think things over before doing anything?	○	○
6.	If you say you will do something do you always keep your promise, no matter how inconvenient it might be to do so?	○	○
7.	Does your mood often go up and down?	○	○
8.	Do you generally do and say things quickly without stopping to think?	○	○
9.	Do you ever feel "just miserable" for no good reason?	○	○
10.	Would you do almost anything for a dare?	○	○
11.	Do you suddenly feel shy when you want to talk to an attractive stranger?	○	○
12.	Once in a while do you lose your temper and get angry?	○	○
13.	Do you often do things on the spur of the moment?	○	○
14.	Do you often worry about things you should not have done or said?	○	○
15.	Generally, do you prefer reading to meeting people?	○	○
16.	Are your feelings rather easily hurt?	○	○
17.	Do you like going out a lot?	○	○
18.	Do you occasionally have thoughts and ideas that you would not like other people to know about?	○	○
19.	Are you sometimes bubbling over with energy and sometimes very sluggish?	○	○
20.	Do you prefer to have few but special friends?	○	○
21.	Do you daydream a lot?	○	○
22.	When people shout at you, do you shout back?	○	○
23.	Are you often troubled about feelings of guilt?	○	○
24.	Are *all* your habits good and desirable ones?	○	○
25.	Can you usually let yourself go and enjoy yourself a lot at a lively party?	○	○
26.	Would you call yourself tense or "highly-strung"?	○	○
27.	Do other people think of you as being very lively?	○	○

		YES	NO
28.	After you have done something important, do you often come away feeling you could have done better?	○	○
29.	Are you mostly quiet when you are with other people?	○	○
30.	Do you sometimes gossip?	○	○
31.	Do ideas run through your head so that you cannot sleep?	○	○
32.	If there is something you want to know about, would you rather look it up in a book than talk to someone about it?	○	○
33.	Do you get palpitations or thumping in your heart?	○	○
34.	Do you like the kind of work that you need to pay close attention to?	○	○
35.	Do you get attacks of shaking or trembling?	○	○
36.	Would you always declare *everything* at the customs, even if you knew that you could never be found out?	○	○
37.	Do you hate being with a crowd who play jokes on one another?	○	○
38.	Are you an irritable person?	○	○
39.	Do you like doing things in which you have to act quickly?	○	○
40.	Do you worry about awful things that might happen?	○	○
41.	Are you slow and unhurried in the way you move?	○	○
42.	Have you ever been late for an appointment or work?	○	○
43.	Do you have many nightmares?	○	○
44.	Do you like talking to people so much that you never miss a chance of talking to a stranger?	○	○
45.	Are you troubled by aches and pains?	○	○
46.	Would you be very unhappy if you could not see lots of people most of the time?	○	○
47.	Would you call yourself a nervous person?	○	○
48.	Of all the people you know, are there some whom you definitely do not like?	○	○
49.	Would you say that you were fairly self-confident?	○	○
50.	Are you easily hurt when people find fault with you or your work?	○	○
51.	Do you find it hard to really enjoy yourself at a lively party?	○	○
52.	Are you troubled with feelings of inferiority?	○	○
53.	Can you easily get some life into a rather dull party?	○	○
54.	Do you sometimes talk about things you know nothing about?	○	○
55.	Do you worry about your health?	○	○
56.	Do you like playing pranks on others?	○	○
57.	Do you suffer from sleeplessness?	○	○

PLEASE CHECK TO SEE THAT YOU HAVE ANSWERED <u>ALL</u> THE QUESTIONS

A Survey of Caffeine Consumption

If you drink coffee, please make the appropriate response.

note 1 cup = 1 breakfast cup

		Yes	No
1.	Do you drink coffee each day?	____	____
2.	What is your average daily intake?		
	(i) 1-3 cups	____	
	(ii) 4-7 cups	____	
	(iii) 7-10 cups	____	
	(iv) More than 10 cups - please specify	____	_____
3.	Please indicate consumption levels for		
	(i) Breakfast	_____	
	(ii) Morning tea	_____	
	(iii) Lunch	_____	
	(iv) Afternoon tea	_____	
	(v) Supper	_____	
		Week	Weekend
4.	Do you consume greater amounts of coffee during t e week or during the weekend?	____	____
5.	What type of coffee do you drink more of		
	(1) Brewed/Perked?	_____	
	(2) Instant?	_____	
	(3) Decaffeinated?	_____	
		Yes	No
6.	Have you ever experienced headache/nausea or when abstaining from coffee?	____	____

7. Do you drink more coffee under different moods?

Yes ____ No ____

Please specify.

_____

_____

Appendix 2

VISUAL ANALOGUE

NAME
SEX
CONDITION

PRE-TEST BLOOD PRESSURE and HEART ATE
(a) systolic BP
(b) diastolic BP
(c) pulse

MOOD SCALE
Indicate your mood by placing a cross (x) at a point on the line which best indicates how you feel now.

Relaxed----------Restless
Normal breathing----------Breathless
Sleepy----------Alert
Unhappy----------Happy
Calm----------Nervous
Confidence lacking----------Confident

VIS"AL ANALOGUE

NAME
SEX
CONDITION

POST-TEST BLOOD PRESSURE and HEART RATE
(a) systolic BP
(b) diastolic BP
(c) pulse

MOOD SCALE
Indicate your mood by placing a cross (x) at a point on the line which best indicates how youfeel now.
Relaxed----------Restless
Normal breathing----------Breathless
Sleepy----------Alert
Unhappy----------Happy
Calm----------Nervous
Confidence lacking----------Confident

Appendix 3

BMDP2V. ANALYSIS ONE

BMDP2V Unequal Cells, INI IAL BLOOD PRESSURES

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PSBP I, PSBP 1, PSBP 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
	INTROEXTRA	1	253.26940	0.76	0.3985
1.	SEX	1	4057.33957	12.15	0.0036
	IS	1	221.64366	0.66	0.4288
	ERROR	14	333.86032		
2.	B (SESSIONS)	2	671.09366	8.81	0.0011
	BI	2	21.71940	0.29	0.7541
	BS	2	4.70185	0.06	0.9403
	BIS	2	258.98840	3.40	0.0477
	ERROR	28	76.17341		

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PDBP I, PDBP 1, PDBP 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
	INTROEXTRA	1	264.70799	0.78	0.3912
1.	SEX	1	849.34737	2.51	0.1353
	IS	1	9.48772	0.03	0.8694
	ERROR	14	338.06667		
2.	B (SESSIONS)	2	701.01881	6.65	0.0043
	BI	2	204.58021	1.94	0.1626
	BS	2	85.18450	0.81	0.4561
	BIS	2	98.74591	0.94	0.4041
	ERROR	28	105.48452		

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PPULSE I, PPULSE 1, PPULSE 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
	INTROEXT	1	747.84600	3.65	0.0769
1.	SEX	1	758.68421	3.70	0.0750
	IS	1	575.52632	2.81	0.1161
	ERROR	14	205.08333		
2.	B (SESSIONS)	2	16.18782	0.20	0.8208
	BI	2	229.28723	2.82	0.0768
	BS	2	120.73363	1.48	0.2442
	BIS	2	220.38275	2.71	0.0842
	ERROR	28	81.40833		

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PMRT I, PMRT 1, PMRT 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	251.09335	4.83	0.0453
	SEX	1	17.52699	0.34	0.5708
	IS	1	19.55655	0.38	0.5496
	ERROR	14	52.01229		
2.	B (SESSIONS)	2	2.08392	0.18	0.8373
	BI	2	84.53813	7.25	0.0029
	BS	2	9.87395	0.85	0.4396
	BIS	2	50.69244	4.35	0.0227
	ERROR	28	11.66353		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PM1 I, PM1 1, PM1 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
	INTROEXT	1	3.24267	1.44	0.2508
1.	SEX	1	9.10247	4.03	0.0644
	IS	1	2.60867	1.15	0.3007
	ERROR	14	2.25898		
2.	B (SESSIONS)	2	1.42711	0.81	0.4542
	BI	2	0.02659	0.02	0.9850
	BS	2	0.71973	0.41	0.6679
	BIS	2	3.97873	2.26	0.1227
	ERROR	28	1.75787		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PM2 I, PM2 1, PM2 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.09488	0.03	0.8726
	SEX	1	3.04661	0.86	0.3705
	IS	1	15.81363	4.44	0.0535
	ERROR	14	3.55888		
2.	B (SESSIONS)	2	2.47022	1.14	0.3345
	BI	2	2.89478	1.33	0.2795
	BS	2	0.59552	0.27	0.7619
	BIS	2	0.55272	0.25	0.7768
	ERROR	28	2.16883		

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PM3 I, PM3 1, PM3 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.59146	0.16	0.6958
	SEX	1	3.40464	0.92	0.3544
	IS	1	4.76464	1.28	0.2762
	ERROR	14	3.71142		
2.	B (SESSIONS)	2	0.17592	0.08	0.9261
	BI	2	0.42914	0.19	0.8298
	BS	2	6.65307	2.91	0.0710
	BIS	2	0.08009	0.04	0.9656
	ERROR	28	2.28556		

ANALYSIS OF VARIANCE FOR 1-ST
DEPENDENT VARIABLE - PM4 I, PM4 1, PM4 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.51655	0.23	0.6420
	SEX	1	1.38486	0.61	0.4495
	IS	1	0.19562	0.09	0.7742
	ERROR	14	2.28749		
2.	B (SESSIONS)	2	1.54373	0.87	0.4294
	BI	2	0.95695	0.54	0.5886
	BS	2	1.16350	0.66	0.5264
	BIS	2	0.26935	0.15	0.8597
	ERROR	28	1.77172		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PM5 I, PM5 1, PM5 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.83034	0.32	0.5778
	SEX	1	0.36625	0.14	0.7108
	IS	1	4.83385	1.89	0.1908
	ERROR	14	2.55735		
2.	B(SESSIONS)	2	0.79054	0.37	0.6928
	BI	2	3.62750	1.71	0.1999
	BS	2	0.25779	0.12	0.8863
	BIS	2	2.40539	1.13	0.3369
	ERROR	28	2.12623		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PM6 I, PM6 1, PM6 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	1.81867	0.72	0.4115
	SEX	1	0.04464	0.02	0.8964
	IS	1	0.66499	0.26	0.6168
	ERROR	14	2.53846		
2.	B(SESSIONS)	2	0.42341	0.41	0.6686
	BI	2	1.58271	1.53	0.2348
	BS	2	0.36306	0.35	0.7076
	BIS	2	0.19183	0.19	0.8321
	ERROR	28	1.03656		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPSBP I, PPSBP1, PPSBP 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.00683	4.99	0.0424
	SEX	1	0.00802	0.60	0.4497
	IS	1	0.01373	0.71	0.4135
	ERROR	14	0.01129	1.22	0.2887
2.	B(SESSIONS)	2	0.05505	4.81	0.0161
	BI	2	0.02570	2.24	0.1248
	BS	2	0.00139	0.12	0.8861
	BIS	2	0.03342	2.92	0.0707
	ERROR	28	0.01146		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPDBP I, PPDBP 1, PPDBP 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.01052	5.80	0.0304
	SEX	1	0.21631	0.31	0.5869
	IS	1	0.02715	6.36	0.0244
	ERROR	14	0.03402	0.80	0.3867
2.	B(SESSIONS)	2	0.11055	3.75	0.0361
	BI	2	0.00631	0.21	0.8088
	BS	2	0.03461	1.17	0.3242
	BIS	2	0.08866	3.01	0.0657
	ERROR	28	0.02950		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PP PULSE I, PP PULSE 1, PP PULSE 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.2249	20.69	0.0005
	SEX	1	0.00216	1.16	0.3000
	IS	1	0.00003	0.11	0.7436
	ERROR	14	0.01942	0.00	0.9717
2.	B(SESSIONS)	2	0.00834	0.34	0.7161
	BI	2	0.06068	2.46	0.1039
	BS	2	0.00567	0.23	0.7964
	BIS	2	0.00848	0.34	0.7123
	ERROR	28	0.02469		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PP MRT I, PP MRT 1, PP MRT 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	1.52388	3.11	0.0998
	SEX	1	1.17591	2.40	0.1438
	IS	1	1.52193	3.10	0.1000
	ERROR	14	0.49046		
2.	B(SESSIONS)	2	1.83226	2.07	0.1456
	BI	2	3.23719	3.65	0.0391
	BS	2	1.86475	2.10	0.1411
	BIS	2	2.06945	2.33	0.1156
	ERROR	28	0.88711		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 1 I, PPM 1 1, PPM 1 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.02001	0.47	0.5049
	SEX	1	2.40152	0.03	0.8689
	IS	1	3.24871	3.39	0.0868
	ERROR	14	0.70794	4.59	0.0502
2.	B (SESSIONS)	2	0.07018	0.11	0.8955
	BI	2	0.18094	0.29	0.7536
	BS	2	0.15032	0.24	0.7902
	BIS	2	0.22775	0.36	0.7011
	ERROR	28	0.63315		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 2 I, PPM 2 1, PPM 2 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.02504	0.04	0.8464
	SEX	1	0.28138	0.44	0.5191
	IS	1	4.97508	7.74	0.0147
	ERROR	14	0.64313		
2.	B(SESSIONS)	2	3.27157	5.01	0.0138
	BI	2	0.50392	0.77	0.4717
	BS	2	0.44347	0.68	0.5151
	BIS	2	0.66060	1.01	0.3764
	ERROR	28	0.65271		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 3 I, PPM 3 1, PPM 3 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.02384	0.01	0.9180
	SEX	1	0.01489	0.01	0.9351
	IS	1	1.39510	0.64	0.4360
	ERROR	14	2.16916		
2.	B(SESSIONS)	2	1.81488	0.68	0.5150
	BI	2	2.57585	0.96	0.3935
	BS	2	1.36200	0.51	0.6060
	BIS	2	2.18922	0.82	0.4509
	ERROR	28	2.67072		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 4 I, PPM 4 1, PPM 4 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.48691	0.61	0.4464
	SEX	1	0.13692	0.17	0.6841
	IS	1	0.15814	0.20	0.6621
	ERROR	14	0.79326		
2.	B(SESSIONS)	2	0.25912	0.96	0.3935
	BI	2	0.53627	2.00	0.1547
	BS	2	0.06901	0.26	0.7753
	BIS	2	0.12421	0.46	0.6345
	ERROR	28	0.26865		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 5 I, PPM 5 1, PPM 5 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.14044	0.06	0.8146
	SEX	1	0.05049	0.02	0.8881
	IS	1	6.67820	2.71	0.1217
	ERROR	14	2.46059		
2.	B (SESSIONS)	2	1.58459	0.76	0.4782
	BI	2	0.2239	0.11	0.8995
	BS	2	0.12182	0.06	0.9435
	BIS	2	3.56208	1.70	0.2005
	ERROR	28	2.09204		

ANALYSIS OF VARIANCE 1-ST
DEPENDENT VARIABLE - PPM 6 I, PPM 6 1, PPM 6 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.28159	0.22	0.6428
	SEX	1	0.66579	0.53	0.4781
	IS	1	1.27669	1.02	0.3300
	ERROR	14	1.25352		
2.	B (SESSIONS)	2	0.11772	0.16	0.8530
	BI	2	0.69335	0.94	0.4020
	BS	2	0.57286	0.78	0.4690
	BIS	2	0.75106	1.02	0.3736
	ERROR	28	0.73624		

Appendix 4

BPDP2V. ANALYSIS TWO

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PSBP 1, PSBP 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	395.69550	0.84	0.3673
	EXPPLAC	1	2123.25106	4.50	0.0424
	IE	1	597.62143	1.27	0.2696
	ERROR	30	472.31058		
2.	B(SESSIONS)	1	481.42884	3.77	0.0616
	BI	1	106.67328	0.84	0.3681
	BE	1	992.58439	7.77	0.0091
	BIE	1	8.28810	0.06	0.8007
	ERROR	30	127.72540		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PDBP 1, PDBP 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.02143	0.00	0.9946
	EXXPLAC	1	666.28810	1.45	0.2382
	IE	1	17.47328	0.04	0.8468
	ERROR	30	460.05503		
2.	B (SESSIONS)	1	1097.71667	7.20	0.0117
	BI	1	36.41296	0.24	0.6286
	BE	1	132.23519	0.87	0.3591
	BIE	1	2.19074	0.01	0.9054
	ERROR	30	154.4444		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPulse 1, PPulse 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	194.75238	0.91	0.3474
	EXPPLAC	1	706.33757	3.30	0.0791
	IE	1	2.75238	0.01	0.9104
	ERROR	30	213.73122		
2.	B(SESSIONS)	1	191.13757	2.01	0.1662
	BI	1	23.49312	0.25	0.6225
	BE	1	22.24868	0.23	0.6318
	BIE	1	21.00423	2.33	0.1375
	ERROR	30	94.91640		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMRT 1, PMRT 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	73.71771	4.54	0.0418
	EXPPLAC	1	35.11094	2.16	0.1524
	IE	1	42.16701	2.59	0.1181
	ERROR	29	16.25411		
2.	B(SESSIONS)	1	120.34508	9.14	0.0052
	BI	1	47.03710	3.57	0.0687
	BE	1	0.16413	0.01	0.9119
	BIE	1	86.25469	6.55	0.0159
	ERROR	29	13.16297		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 1 1, PMOOD 1 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.12202	0.04	0.8384
	EXXPLAC	1	0.00536	0.00	0.9656
	IE	1	4.03499	1.42	0.2424
	ERROR	30	2.83687		
2.	B(SESSIONS)	1	1.06673	0.48	0.4948
	BI	1	0.54288	0.24	0.6256
	BE	1	0.00688	0.00	0.9561
	BIE	1	7.63651	3.42	0.0743
	ERROR	30	2.23299		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 2 1, PMOOD 2 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.20007	0.06	0.8121
	EXPPLAC	1	2.08140	0.60	0.4452
	IE	1	0.07029	0.02	0.8879
	ERROR	30	3.47796		
2.	B (SESSIONS)	1	3.68984	2.05	0.1625
	BI	1	7.63651	4.24	0.0482
	BE	1	0.20007	0.11	0.7411
	BIE	1	0.38807	0.22	0.6457
	ERROR	30	1.79951		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 3 1, PMOOD 3 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	4.52610	1.18	0.2853
	EXPPLAC	1	0.08010	0.02	0.8859
	IE	1	7.89943	2.07	0.1610
	ERROR	30	3.82462		
2.	B (SESSIONS)	1	0.68271	0.29	0.5939
	BI	1	0.04471	0.02	0.8912
	BE	1	11.00952	4.68	0.0385
	BIE	1	0.00382	0.00	0.9681
	ERROR	30	2.35090		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 4 1, PMOOD 4 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.00076	0.00	0.9856
	EXPPLAC	1	0.35632	0.15	0.6973
	IE	1	0.82076	0.36	0.5556
	ERROR	30	2.31016		
2.	B (SESSIONS)	1	1.20536	1.07	0.3092
	BI	1	0.03143	0.03	0.8685
	BE	1	2.07202	1.84	0.1852
	BIE	1	2.62965	2.33	0.1370
	ERROR	30	1.12653		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 5 1, PMOOD 5 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
	INTROEXT	1	5.08934	1.44	0.2390
	EXPPLAC	1	0.07378	0.02	0.8859
	IE	1	5.08933	1.44	0.2390
	ERROR	30	3.52562		
2.	B (SESSIONS)	1	8.41029	3.83	0.0598
	BI	1	1.21251	0.55	0.4634
	BE	1	0.97473	0.44	0.5105
	BIE	1	5.43251	2.47	0.1264
	ERROR	30	2.19796		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PMOOD 6 1, PMOOD 6 2.

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	2.91667	1.30	0.2636
	EXPPLAC	1	0.82963	0.37	0.5480
	IE	1	1.00385	0.45	0.5090
	ERROR	30	2.24752		
2.	B (SESSIONS)	1	3.74630	3.48	0.0721
	BI	1	0.27430	0.25	0.6176
	BE	1	2.54074	2.36	0.1351
	BIE	1	1.62607	1.51	0.2288
	ERROR	30	1.07759		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPSBP1, PPSBP2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.00064	0.09	0.7652
	EXPPLAC	1	0.00018	0.03	0.8729
	IE	1	0.03242	4.63	0.0395
	ERROR	30	0.00700		
2.	B (SESSIONS)	1	0.03160	2.04	0.1632
	BI	1	0.00230	0.15	0.7024
	BE	1	0.03160	2.04	0.1632
	BIE	1	0.00013	0.01	0.9287
	ERROR	30	0.01547		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPDBP 1, PPDBP 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PRO-BABILITY
1.	INTROEXT	1	0.10434	1.86	0.1827
	EXPPLAC	1	0.00000	0.00	0.9989
	IE	1	0.03499	0.62	0.4358
	ERROR	30	0.05608		
2.	B(SESSIONS)	1	0.3721	0.74	0.3964
	BI	1	0.00946	0.19	0.6675
	BE	1	0.00084	0.02	0.8981
	BIE	1	0.01585	0.32	0.5786
	ERROR	30	0.05027		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PP PULSE 1, PP PULSE 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.01788	1.32	0.2596
	EXXPLAC	1	0.05053	3.73	0.0629
	IE	1	0.01334	0.99	0.3289
	ERROR	30	0.01354		
2.	B (SESSIONS)	1	0.00241	0.19	0.6623
	BI	1	0.03797	3.06	0.0904
	BE	1	0.00812	0.65	0.4248
	BIE	1	0.00339	0.27	0.6049
	ERROR	30	0.01240		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PP MRT 1, PP MRT 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.05496	1.49	0.2326
	EXPPLAC	1	0.18110	4.90	0.0349
	IE	1	0.04317	1.17	0.2888
	ERROR	30	0.03698		
2.	B (SESSIONS)	1	0.09958	1.97	0.1708
	BI	1	0.00178	0.04	0.8523
	BE	1	0.06760	1.34	0.2567
	BIE	1	0.00008	0.00	0.9687
	ERROR	30	0.05049		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE PP MOOD 1 1, PP MOOD 1 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.06016	0.09	0.7697
	EXXPLAC	1	0.03426	0.05	0.8251
	IE	1	0.63053	0.91	0.3466
	ERROR	30	0.68956		
2.	B(SESSIONS)	1	0.03963	0.11	0.7461
	BI	1	0.07290	0.20	0.6608
	BE	1	0.02656	0.07	0.7909
	BIE	1	0.49326	1.33	0.2581
	ERROR	30	0.37117		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPMOOD 2 1, PP MOOD 2 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.78780	0.14	0.7148
	EXXPLAC	1	7.68243	1.33	0.2584
	IE	1	0.28827	0.05	0.8249
	ERROR	30	5.78773		
2.	B(SESSIONS)	1	0.21413	0.05	0.8208
	BI	1	11.88210	2.90	0.0991
	BE	1	0.05913	0.01	0.9052
	BIE	1	12.58287	3.07	0.0901
	ERROR	30	4.10201		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPMOOD 3 1, PP MOOD 3 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	9.55328	2.04	0.1630
	EXXPLAC	1	3.47499	0.74	0.3953
	IE	1	1.66488	0.36	0.5550
	ERROR	30	4.67169		
2.	B (SESSIONS)	1	3.81143	0.92	0.3463
	BI	1	4.44000	1.07	0.3100
	BE	1	2.24939	0.54	0.4680
	BIE	1	2.76805	0.66	0.4213
	ERROR	30	4.16327		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPMOOD 4 1, PPMOOD 4 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.00084	0.00	0.9616
	EXPPLAC	1	0.48326	1.36	0.2524
	IE	1	0.09212	0.26	0.6142
	ERROR	30	0.35491		
2.	B (SESSIONS)	1	0.03178	0.11	0.7437
	BI	1	0.24707	0.85	0.3648
	BE	1	0.14585	0.50	0.4850
	BIE	1	0.19861	0.68	0.4158
	ERROR	30	0.29172		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPMOOD 5 1, PPMOOD 5 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.34802	0.12	0.7362
	EXPPLAC	1	0.15175	0.05	0.8238
	IE	1	2.79301	0.93	0.3430
	ERROR	30	3.00911		
2.	B(SESSIONS)	1	0.02443	0.01	0.9139
	BI	1	3.20630	1.56	0.2215
	BE	1	1.20643	0.59	0.4497
	BIE	1	0.87437	0.43	0.5193
	ERROR	30	2.05657		

ANALYSIS OF VARIANCE 1ST
DEPENDENT VARIABLE - PPMOOD 6 1, PPMOOD 6 2

	SOURCE	DEGREES OF FREEDOM	MEAN SQUARE	F	TAIL PROBABILITY
1.	INTROEXT	1	0.93469	0.63	0.4330
	EXPPLAC	1	1.58814	1.07	0.3085
	IE	1	1.02878	0.70	0.4110
	ERROR	30	1.47997		
2.	B(SESSIONS)	1	0.38483	0.28	0.5977
	BI	1	0.77429	0.57	0.4552
	BE	1	2.31079	1.71	0.2011
	BIE	1	2.06828	1.53	0.2258
	ERROR	30	1.35266		